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Ecological and Human Health Risk Assessment Guidance for Aquatic Environments

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Dredging: Contaminated Sediments

Ecological and Human Health Risk Assessment Guidance for Aquatic Environments (TR DOER-4)

ISSUE: Evaluating the potential environmental consequences associated with dredging and dredged material disposal is a challenging task. Scientific advancements have made possible the collection of large amounts of complex technical information. The dredged material manager must often weigh and balance multiple and sometimes conflicting lines of evidence to reach a decision; and each decision involves a certain level of uncertainty. The application of Environmental Risk Assessment methods will increase a manager's ability to make objective management decisions when data collected in Tiers I-III of the dredged material evaluation framework are insufficient for decision making.

RESEARCH: The objective was to develop guidance for conducting human health and ecological risk assessments to evaluate potential impacts associated with aquatic placement of dredged material.

SUMMARY: The guidance contained within this report includes an overview of ecological and human health risk assessment and recommendations on the proper application of risk assessment within the dredging program. Guidance for assessing ecological risk includes a discussion of problem formulation, including conceptual model development and the selection of assessment and measurement endpoints, exposure and effects assessment, and risk characterization. Standard approaches for assessing human health risk, including hazard identification, toxicity assessment, and risk characterization, are also discussed within the context of aquatic placement of dredged material. Guidance is provided for conducting uncertainty analysis for both ecological and human health risk assessments. Sources of additional information on risk assessment applications, toxicity profiles, and other tools used in risk assessment are provided in appendixes.

AVAILABILITY OF REPORT: The report is available in .pdf format on the World Wide Web at *http://www.wes.army.mil/el/dots* and through Interlibrary Loan Service from the U.S. Army Engineer Research and Development Center (ERDC), Waterways Experiment Station (WES) Library, telephone (601) 634-2355.

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Preface

The work reported herein was conducted in the Risk Focus Area of the Dredging Operations Environmental Research (DOER) Program, Environmental Laboratory (EL), Waterways Experiment Station (WES), Vicksburg, MS, U.S. Army Engineer Research and Development Center (ERDC). The DOER Program is sponsored by Headquarters, U.S. Army Corps of Engineers. The program managers for the DOER program are Dr. Robert Engler and Mr. Clark McNair, EL. Technical Monitor for this study was Mr. Joe Wilson.

The WES principal investigators for this work were Drs. Todd S. Bridges and David W. Moore. This report was authored by Drs. Jerome J. Cura and Wendy Heiger-Bernays, Menzie-Cura & Associates, Inc.; Drs. Bridges and Moore.

Special thanks go to the following Corps of Engineers personnel who gave generously of their time and expertise to review the drafts: Mr. Joseph Wilson, whose comments encouraged the authors to produce a clear and utilitarian document consistent with the application of risk assessment to dredged material management; Dr. Engler, for his willingness to share his wealth of experience and his insistence on explicit language and clarity; and Dr. Victor McFarland, for his careful review of ecological toxicity; Dr. Michael Palermo, for his comments concerning fate and transport and engineering alternatives. We gratefully acknowledge the technical review of an earlier version of the document by United States Environmental Protection Agency personnel, Drs. Philip Cook, Norman Rubinstein, and Richard Pruell.

At the time of preparation of this report, Dr. Ed Link was Acting Director of ERDC, and COL Robin R. Cababa, EN, was Commander.

Ecological and Human Health Risk Assessment Guidance

What Is the Purpose of this Document?

This document provides guidance for conducting ecological and human health risk assessments at aquatic sites potentially impacted by dredged material management activities.

What is Risk Assessment?

Risk assessment is the process of evaluating the impact of a chemical or physical condition upon the health of individual humans or the environmental well-being of a population or community of animals and plants. The former is *called human health risk assessment,* and the latter *ecological risk assessment.*

Proper Timing for the Risk Assessment Option?

The project manager should decide to apply a risk assessment within the context of the site selection process and/or the four-tiered evaluation of dredged material, or when there are unresolved issues with regard to potential human or ecological exposures. It is most applicable to projects which have:

a. Reached Tier IV and concern about specific bioaccumulative compounds or toxic compounds remains.

b. The potential to affect a local sensitive habitat or species.

c Outstanding exposure issues where a risk assessment will allow realistic use of information about a species' natural history such as foraging areas, breeding times, migration patterns.

d. Potential human health exposure either directly to sediments or through the food chain.

e. Issues associated with environmental windows (time periods when a species is least vulnerable).

Who Can Conduct a Risk Assessment?

The selection of personnel to conduct a risk assessment depends on the level of complexity addressed in the risk assessment. For example, a rough estimate of exposure based on a simple sediment-water partitioning equation may be sufficient to demonstrate little probability of bioavailability of a chemical, and hence risk. In such a case, operations personnel with expertise in engineering, chemistry, or marine geology may be the only necessary personnel. In the most complex assessments (and these are likely to be the least frequently encountered), an interdisciplinary team of engineers, biologists, chemists, and physical scientists may be necessary.

1 Overview of Ecological and Human Health Risk Assessment Guidance for Dredged Material Management

Purpose and Organization

Purpose

This document provides guidance to United States Army Corps of Engineers (USACE) project managers and field operations personnel in the use of risk assessment to facilitate dredged material management decision-making. It specifically addresses the management of dredged material in an aquatic environment. It does not address risk associated with the management of dredged material in upland environments. Also, the document addresses only chemical contamination and does not address other potential sources of impact such as physical disturbance. The intended audience and user community are the individual scientists and managers making decisions where there are competing interests on the dredging and disposal management of sediments from the nation's waterways.

The document does not promote risk assessment as a tool for use in every dredged material management decision. It is likely to be most useful, and most used, in those cases which constitute the exception rather than the rule. The use of risk assessment is intended to supplement the analytical options currently available to dredged material managers by building on the existing technical framework United States Environmental Protection Agency (USEPA)/USACE 1992) and the existing tiered sediment evaluation approaches (USEPA/USACE 1991, 1998).

Scientific advancements have made possible the collection of large amounts of complex information regarding the environmental aspects of dredging and dredged material disposal. The dredged material manager must often use "best professional judgement" to weigh and balance among multiple and sometimes conflicting lines of evidence to reach a decision. Environmental risk assessment provides a stepwise framework for the integration of complex information to yield quantifiable estimates of risk including uncertainty. In addition, risk assessment

allows the dredged material manager to make explicit the types of information considered and how a decision is reached regarding the suitability of a dredged material for a particular management option.

Organization of the document

This document describes the various components of risk assessments including:

This Overview, which provides an overview of the various elements in risk assessment, the relationship of risk assessment to the tiered sediment evaluation procedures, and the relationship between ecological and human health risk assessment.

Section 2, Problem Formulation, which describes the objectives of risk assessment, development of a site conceptual model, selection of contaminants of concern, a procedure for selecting the organisms and humans of concern at a dredged material management site, and a method for deciding on decision criteria (endpoints) for the risk assessment.

Section 3, Ecological Exposure Assessment, Effects Assessment, and Risk Characterization, which describes how to estimate ecological exposure to contaminants of concern and characterize risk from such exposures.

Section 4, Human Health Risk Assessment, which describes how to estimate human exposure to contaminants of concern and characterize risk from such exposures.

Appendix A, Summary of Federal, Regional, and State Guidance, which reviews available Federal, regional, and state guidance and methods used by human health and ecological risk assessors.

Appendix B, Information Sources, which describes the content and availability of various text and on-line information important in conducting risk assessments.

Appendix C, Food Chain and Toxicity Models, which describes some food chain models useful in risk assessment.

Appendix D, Toxicological Profiles, which provides toxicological profiles (i.e., summaries) for the likely contaminants of concern at dredged material management sites.

Appendix E, Human Health Exposure Equations, which provides detailed human health exposure equations for various potential human exposure scenarios at dredged material management sites.

Appendix F, A Hypothetical Example, which illustrates the major points in the guidance. Each section presents the guidance as a continuous example in a series of "Example Boxes" numbered sequentially within each chapter. The hypothetical

example provides a continuous example in uninterrupted text, for the reader's convenience.

Appendix G is a Glossary of Terms. Often, and contrary to USEPA directive to be transparent, discussion of risk assessment is obfuscated with technical jargon and "terms of art." This appendix attempts to provide definitions for such terms in simple business English and emphasizes the initial use of the term in bold italics.

Background

The USACE navigation mission entails maintenance and improvement of 40,225 km of channels, supporting a vital component of the Nation's transportation infrastructure system. These waterways serve 400 ports, including 130 of the Nation's 150 largest cities

The USACE dredge and/or permit for dredging an annual average of 191 to 229 million cu m of sediment from this navigation system at an annual cost of $400 to $600 million. Dredging is the single most costly item in the Corps' Civil Works budget. Corps grants are also permitted to the private sector for dredging and disposing of an additional 764,600 cu m of sediment.

These dredged sediments, especially in urbanized and industrial harbors, may exhibit high concentrations of various contaminants from years of unregulated discharge and runoff. Selecting appropriate management options for contaminated sediment is a difficult task, exacerbated by the rapidly diminishing capacity of existing management locations and by public resistance to construction of new facilities in traditional locations. Management options are quickly disappearing, and the seasonal periods available for dredging are increasingly constrained by environmental windows and other restrictions for the protection of sensitive aquatic resources and wildlife.

Today's dredging manager faces a complex situation requiring a cost-efficient operation which simultaneously considers the risks associated with various types of dredging equipment, timing of dredging and management operations, selection of an appropriate management alternative, and determining the relative importance of ecological impacts from the management operation.

Fiscal constraints add further difficulty to a district's maintenance dredging/management program. The use of risk management can facilitate the efficient use of limited funds through evaluation of critical factors (e.g., cost, equipment, windows, contaminants, disposal options, shoaling and channel navigability, etc.) as well as the consequence of not dredging. This document develops a repeatable and defendable framework to assess the risks from exposure to contaminants in aquatic systems associated with management options.

What is Risk Assessment?

Risk assessment is the process of evaluating the impact of a stressor (e.g., a chemical or physical condition) upon the health of individual humans or the environmental well-being of a population or community of animals and plants.

The former is called human health risk assessment, and the latter is called ecological risk assessment. Subsequent sections describe how these two categories of risk assessment differ.

Risk assessment in its more common manifestations is an often used, although not necessarily formally recognized, component of the dredged material management decision-making process. For example, Peddicord et al. (1997) note that the present procedure for evaluating water column impacts in dredged material evaluations (USEPA/USACE 1991, 1998) is an application of ecological risk assessment.

In its most basic form, risk assessment means answering several simple questions which usually underlie dredged material management decisions. These include:

a. Are there humans, organisms, or habitats (all called receptors) near the proposed dredged material management activities?

b. Are there chemicals or physical hazards associated with the proposed dredged material which may affect the survival or reproduction of these receptors? The answer to this question is called a hazard identification.

c. Is there a known quantity of the chemical or physical hazard which results in an adverse effect to the likely receptors? This is called toxicity assessment or effects assessment.

d. Are there any conservative, but realistic, activities or physical and biological pathways by which the receptors may encounter the chemical or physical hazards associated with a particular proposed dredged material activity? This is termed exposure assessment.

e. Finally, under a specified set of conditions, will this encounter result in an exposure to the chemical or physical hazard at a level known to cause an adverse effect? (Risk characterization).

Generally, if the answer to this last question is no, then we assume that the risk associated with the dredged material management decision is acceptable. If it is yes, then there is some potential unacceptable risk, and we begin to search for ways to modify management activities or receptor activities to lower the exposure and hence risk. The decision maker asks one additional question:

f. How confident are we in our answer? (Uncertainty analysis).

Viewed as a formal approach to answering these simple and commonly posed questions, risk assessment appears as a familiar thought process. Also, dredged material managers and USACE field operations personnel will recognize that the information necessary to answer these questions is nearly always available from data developed as part of the site selection process and tiered evaluation process described in the Dredged Material Testing Manuals (USEPA/USACE 1991, 1998).

A risk assessment is essentially complete when it provides defensible answers to the above questions. Current Federal, state, and industry guidance recognizes that risk assessment can be a fairly simple set of answers to these questions. The level of effort needed ranges from a simple "back of the envelope" calculation to something as sophisticated as integrating the various fate and transport models available from U.S. Army Engineer Waterways Experiment Station (WES) (e.g., ADDAMS. See USACE 1995a) with one of several biological food chain models available in the scientific literature. The Corps is preparing a series of technical documents which will guide managers and operations personnel in the appropriate application of these models. The Corps is also developing a series of technical guidance and support documents and on-line databases to support field operations personnel in conducting risk assessment.

Proper Timing for Risk Assessment

The project manager should decide to apply a risk assessment within the context of the site selection process and/or the tiered evaluation of dredged material, or when there are unresolved issues with regard to potential human or ecological exposures. Risk assessment is not separate from the current methods of decision-making. It merely enhances them.

A formal assessment is not something to be applied to every project. It is most applicable to projects which have:

a. Reached Tier IV and concern about specific bioaccumulative compounds or toxic compounds remains.

b. The potential to affect a local sensitive habitat or species.

c. Outstanding exposure issues where a risk assessment will allow realistic use of information about a species' natural history such as foraging areas, breeding times, migration patterns.

d. Potential human health exposure either directly to sediments or through the food chain.

e. Issues associated with environmental windows.

Risk assessment is not applied to the typical dredged material site or project which is easily handled through the existing technical framework. Rather, it applies in those cases where an extended analysis allows the dredged material manager to address such real-world conditions as sediment matrix effects, bioavailability, intermittent use of the site by a species of concern, the mitigating effects of a specific management technology, the likely exposure to people fishing recreationally, etc.

Risk Assessment Role in Dredged Material Risk Management Process

Risk assessment alone cannot compel a decision at a dredged material management site. In those cases where the dredged material manager chooses to apply risk assessment, he or she should consider it as part of a larger risk analysis process which includes risk management. In prior considerations of risk management, the USACE (1995b) views this process as a function of several factors: risk and uncertainty, cost, schedule, value of resources protected, regulatory requirements, political, economic, technical feasibility, environmental justice/equity. The role of the risk assessment in this general process is to provide realistic assessments, not hypothetical or highly conservative assessments that provide no meaningful risk information to decision makers. Within the risk management process, the risk assessment contributes most readily to the evaluation of alternatives.

The Framework Document (USEPA/USACE 1992) provides comprehensive guidance on identifying, screening, and selecting "reasonable" dredged material disposal alternatives. The primary, although not exclusive, considerations when evaluating disposal alternatives are effectiveness, implementability, and cost.

Risk Assessment Format

There are numerous program-specific documents which describe the formal components of a risk assessment and details of conducting assessment within the constraints of the program. The dredged material manager should recognize that there are several general components included in risk assessments, based on an USEPA framework (USEPA 1992a) and recently published USEPA guidelines USEPA 1998). These components address the initial questions indicated earlier. The risk assessment process has five general components (Figure 1).

a. *Hazard identification/problem formulation.* Hazard identification is the process of determining whether exposure to a contaminant can cause an increase in the incidence of a particular human health (e.g., cancer, birth defect, etc.) or ecological (e.g., reproductive, lethal, etc.) effect. In ecological risk assessment, the selection of receptors begins in this section, but is a process which will continue into the Exposure Assessment.

b. *Exposure assessment.* An exposure assessment estimates the magnitude of actual and/or potential human or ecological exposure to a contaminant of concern, the frequency and duration of exposure, and the pathways of exposure for human and ecological receptors. This is the major step in the development of scenarios, and the decisions made during the exposure assessment will be critical to the ultimate estimate of risk. To address concerns of stakeholders, it is important that this aspect of scenario development be a cooperative effort early in the risk assessment process. An important component of exposure assessment is the selection of

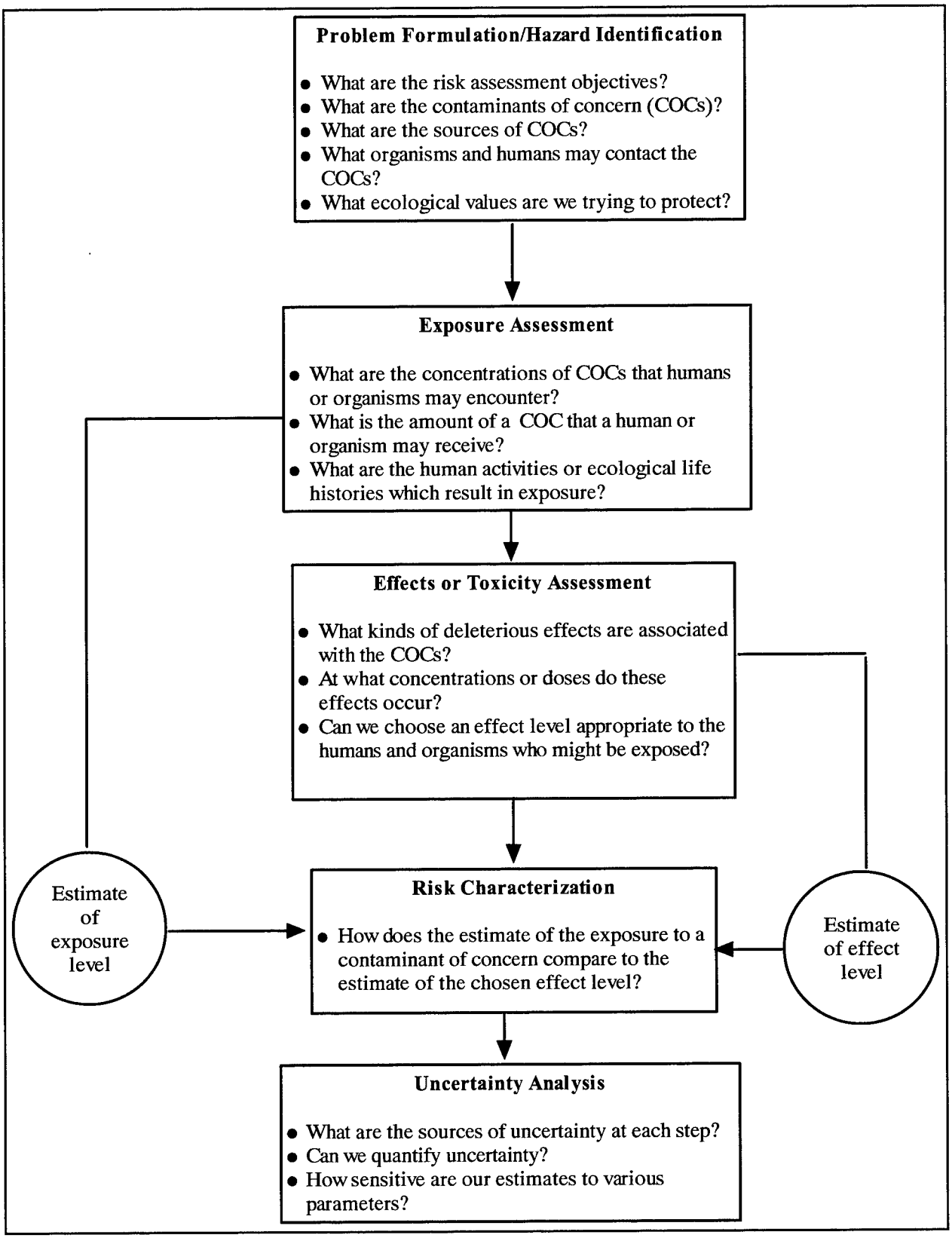

Figure 1. Components of risk assessment and questions addressed

human and ecological receptors. To a large extent, these will drive the development of exposure pathways.

c. *Toxicity assessment/effects assessment.* The toxicity assessment summarizes and weighs available evidence regarding the potential for contaminants to cause adverse effects in exposed individuals and to provide, where possible, an estimate of the relationship between the extent of exposure to a contaminant and the increased likelihood and/or severity of adverse effects. Current guidance for ecological risk assessment often refers to "toxicity assessment" as an "effects

d. *Risk characterization.* The risk characterization summarizes and integrates the exposure assessment and toxicity assessment into a quantitative and qualitative expression of risk. In a human-health risk assessment, the risk characterization:

   (1) Characterizes carcinogenic effects by estimating probabilities that an individual will develop cancer over a lifetime of exposure based on projected intakes from a given scenario and the information summarized in the toxicity assessment.

   (2) Characterizes noncarcinogenic effects by comparing calculated intakes of substances, based on specific exposure scenarios, to acceptable doses.

   Generally in an ecological risk assessment, risk characterization evaluates risk by comparing a concentration, dose, or body burden known to produce an effect, with a corresponding measurement or projection of exposure made in the exposure assessment (*toxicity quotient method*). The risk assessor may consider the toxicity quotient with other sources of information (biological conditions at the site, information from reference areas) to form a professional opinion regarding potential risk in a *weight of evidence approach.*

e. *Uncertainty analysis.* The risk characterization should also address uncertainty in the analysis of human health and ecological risk. Risk assessments do not generally provide fully probabilistic estimations of risk. Therefore, highly quantitative statistical uncertainty analyses are not common. USEPA/OERR (1989a) indicates the importance of identifying the key site-related variables and assumptions that contribute most to the uncertainty.

Ecological and Human Health Risk Assessment Relationship

At most sites, risk assessment will address two general types of risk, ecological risk and human health risk. Ecological risk assessment focuses on potential risk to nonhuman biota likely to occur at a disposal site. Human health risk assessment focuses on carcinogenic and noncarcinogenic risk to humans from potential exposure. A major difference between the two is that a human health risk

assessment addresses potential effects to one type of receptor, human beings, while ecological risk assessment can address risk to several receptors chosen to represent the ecosystem associated with the dredged material disposal site.

These two types of risk assessment address the fate and transport of contaminants in similar, if not identical manners. Those physical and chemical processes which drive the distribution of contaminants will not change between the two types of risk assessment. The two are linked in that the estimates of contaminant uptake by biota (evaluated in the ecological risk assessment) may result in exposure to humans if people eat that organism. Clearly, the feeding habits of a commercial species, an ecological characteristic, will to a large extent determine whether that species can pass a contaminant on to a human. This is the point where ecological and human health risks are most closely linked. They diverge in the discussion of toxicological processes and how these processes relate to potential effects.

Who Can Conduct a Risk Assessment?

The selection of personnel to conduct a risk assessment depends on the level of complexity addressed in the risk assessment. For example, a rough estimate of exposure based on a simple sediment-water partitioning equation may be sufficient to demonstrate little probability of bioavailability of a chemical and, hence, risk. In such a case, operations personnel with expertise in engineering, chemistry, or marine geology may be the only necessary personnel. In the most complex assessments (and these are likely to be the least frequently encountered), an interdisciplinary team of engineers, biologists, chemists, and physical scientists may be necessary.

Data Collection Requirements of Risk Assessment

The site selection process and the dredged material evaluation tiered approach will satisfy most risk assessment data needs (Table 1). These data may have to be reformulated to provide direct answers to the six questions posed earlier.

The initial question, "Are there humans, organisms, or habitats near the proposed dredged material management activities?", is usually directly answered in the baseline studies of the site selection process. These studies generally define and describe sensitive habitats or species, commercially important species using the site, recreational or commercial uses of the site, and the types of biological communities nearby. Risk assessment may require some reformulation or expansion of this information, if an analysis of potential exposure pathways reveals data gaps. For example, a risk assessment may require a more detailed description of human use of the site or an expansion of species descriptions to include information on life history. Usually such can be satisfied by an expanded literature review.

The dredged material evaluation will provide the necessary data to address the Hazard Identification question, "Are there stressors associated with a proposed management action which may affect the survival or reproduction of these receptors?" The Tier I characterization of the sediments relies on available results

Table 1.
Information Sources for Risk Assessment Within the Dredged Material Management Program
("✓ " Indicates information is available for use in a particular section of risk assessment)

	Site Selection Report and Associated Environmental Reports	Tier I	Tier II	Tier III	Tier IV
Type of Information Available	Sensitive habitats or species, commercially important species using the site, recreational or commercial uses of the site, types of biological communities nearby	Characterize sediment; selection of COCs; review existing data	Predictive models to assess physical transport and water quality impacts; Theoretical Bioaccumulation Model	Water column toxicity; sediment toxicity; bioaccumulation testing	Chronic sublethal sediment toxicity; steady-state bioaccumulation
Risk Assessment Component					
Identify Receptors	✓				
Hazard Identification		✓			
Identify COCs		✓			
Toxicity Assessment		✓	✓	✓	✓
Exposure Assessment	✓	✓	✓	✓	✓
Risk Characterization		✓		✓	
Uncertainty	✓	✓	✓	✓	✓

of prior chemical testing, measurements of physical characteristics, organic carbon content, grain size, and review of regulatory records and published literature regarding the material to be dredged (published studies, permit reviews, federal databases, etc.). This information is generally sufficient for a risk assessor to develop the Hazard Identification and develop a list of *contaminants of concern* (COCs). Note that specifying COCs is an integral part of risk assessment which will have already been accomplished as a Tier I activity based on explicit criteria in the several dredged material testing manuals (USEPA/USACE 1991, 1998).

The identification of COCs during Tier I depends in part on the toxicological importance of each contaminant. This Tier I task therefore provides a start on the risk assessment's Toxicity or Effects Assessment which answers the question, "Is there a known quantity of the chemical or physical hazard which results in an adverse effect to the likely receptors?" The risk assessment may require that this information be reformulated to conform to the parameters used in human health or ecological exposure models. This is generally accomplished by reference to on-line USEPA and USACE databases or an expanded literature review.

The exposure assessment addresses the question, "Are there any conservative but realistic, activities or physical and biological pathways by which the receptors may encounter the chemical or physical hazards?" This is a considerable expansion of Tier I sediment characterizations or Tier II modeling activities and also incorporates the bioaccumulation testing conducted in Tier III. This is the risk assessment component which will require the most expansion upon prior data gathering activities because this is the point which integrates the site selection information with the dredged material evaluation. Although it generally will not require new data collection, it will require a reformulation of the information into a site-specific conceptual model.

In summary, the activities of site selection and dredged material evaluation provide most of the information needed to conduct a risk assessment. There will be some necessary renewed literature reviews and a reformulation of the data, but expensive, time-consuming field data collections are unlikely.

Problem Formulation

What is problem formulation?

The *problem formulation* of a risk assessment is a systematic planning stage that identifies the major factors considered in the assessment, and establishes its goals, breadth, and focus (USEPA/ Environmental Response Team (ERT) 1997). (Note that in human health risk assessment, this stage is called hazard identification). This step requires reviewing and summarizing information on the management activities, likely contaminants, the environmental setting, the human uses of the area, and its resources.

What occurs in problem formulation?

Four major activities occur during the problem formulation:

a. Developing the objectives of the risk assessment - stating clearly what the specific risk assessment should accomplish.

b. Developing a Conceptual Model - to a large degree this is a qualitative analysis in narrative and graphical format of how contaminants from dredged material management activities may be reaching humans or organisms.

c. Selecting and Characterizing Receptors - selecting and describing organisms and humans which best represent the types of organisms and human activities that may contact contaminants from the dredged material management site.

d. Developing Endpoints - describing what environmental resources the risk assessment is trying to protect and what measurements will be used to assess whether that resource is at risk (note that human health risk assessment endpoints are explicitly set by convention).

2 Problem Formulation

The problem formulation of a risk assessment is a systematic planning stage that identifies the major factors considered in the assessment, and establishes its goals, breadth, and focus (USEPA/ERT 1997). It is essentially a scoping activity and is fundamental to the success of all subsequent components in the risk assessment. There are four general activities within problem formulation.

a. Statement of objectives: The risk manager initiates the problem formulation with a statement of objectives. Subsequent selection of assessment techniques and procedures largely depends on this objective statement. Consequently, time spent by the dredged material manager in addressing why the risk assessment is being performed will substantially improve the decision-making process.

b. Development of a conceptual model: The conceptual model specifies the pathways by which a contaminant of concern might move from the management area to a human or organism of concern.

c. Selection and characterization of receptors: This task selects and describes organisms and humans which best represent the types of organisms and human activities that may contact contaminants from the dredged material management site.

d. Identifying endpoints: The human health risk assessment has numerical endpoints specified by convention to protect humans against carcinogenic and noncarcinogenic health risks. However, for ecological receptors, the risk assessment will use endpoints which depend upon the ecological characteristics of the management area and management activity under consideration. Assessment endpoints are the valued characteristics of a management site or adjacent ecosystem that should be protected. In selecting appropriate assessment endpoints, some factors to be considered include the ecological relevance of the endpoint, policy goals and societal values, and susceptibility to the contaminant. Measurement endpoints are discrete observations that can be related to the assessment endpoint. Generally, we must extrapolate from the measurement endpoints back to the assessment endpoints in judging whether the value expressed by the assessment endpoint is at risk.

Objectives of Ecological Risk Assessment

Each site-specific ecological risk assessment should have its own set of objectives. Many of these may be associated with specific issues, unique to a given site. It is important for the risk assessor to specify any site-specific objectives in advance of subsequent analyses. Obviously, this process is iterative. Site-specific objectives may become sharper, or even modified, as the analyses progress. In addition, site-specific objectives should be agreed upon "up-front" based on input from dredged material managers, stakeholders, and environmental groups.

There are several objectives common to all risk assessments: These include:

a. Identify contaminants of concern.

b. Identify organisms, ecosystems, and people that may be exposed to contaminants contained in the dredged material.

c. Select organisms and humans which represent the ecosystem and human activities associated with the dredged material site.

d. Identify the pathways by which receptors may be exposed to the contaminants.

e. Specify the valued characteristics of the exposed organisms or ecosystem.

f. Specify measured or estimated concentrations of contaminants of concern which organisms or humans may contact.

g. Develop information on the toxic effects of contaminants of concern.

h. Characterize the ecological and human risks associated with the exposure under current and future conditions.

i. Assess the uncertainties associated with measurements, estimates, and risk characterizations.

There may be other site-specific objectives raised by local groups or regulators. The risk assessment should incorporate these into a statement of objectives.

The product of this section of the ecological risk assessment will be a clearly written set of objectives which will reflect the concerns of interested parties. These concerns and how the objectives relate to them should be in the written document. These objectives will guide the remaining steps in the ecological risk assessment.

Developing a Conceptual Model

What is Purpose of the Conceptual Model?

This section provides guidance for developing a *conceptual model* by asking these simple questions:

a. What humans or other organisms might be exposed to contaminants associated with dredged material management activities?

b. What are the contaminants associated with the dredged material?

c. What are the physical or biological processes which might link the contaminants with the humans or other organisms?

The development of the conceptual model poses these questions and takes the initial steps toward answering them. However, this attempt is the overall task of the risk assessment which will revisit these questions in an iterative manner throughout the process.

How Does the Risk Assessment Develop the Answers to These Questions?

As the first step in an iterative process, the conceptual model is an integration of existing information in a graphical and written format. The level of detail will vary with the complexity of the local environment, the number and types of contaminants, and the various dredged material management alternatives under consideration.

The development of the conceptual model requires characterizing the environmental setting and describing the potentially complete exposure pathways. The dredged material manager will recognize that much of the information necessary to develop the conceptual model is available through the Tiered Evaluation Process.

Development of a Conceptual Model

The term conceptual model is a "term of art" in risk assessment and has specific meaning. The conceptual model is an integration of existing information which attempts to identify the contaminants and their sources, describe the pathways by which they may reach humans or other organisms, and specify which humans or organisms might be linked to the contaminants by these pathways. These humans and organisms are called receptors. The assessment presents the conceptual model as a narrative or diagram which describes the links between contaminant sources and receptors along explicit fate and transport pathways. As demonstrated in the various summaries of state, Federal, and industry guidance in Appendix A, nearly all guidance documents for risk assessments require the development of a conceptual model.

The development of the conceptual model may resolve questions. For example, any incomplete exposure pathways defined in the conceptual model are eliminated from further consideration. This is the opportunity to focus the questions upon those issues of real concern. In the development of the conceptual model, it is important, to obtain meaningful information through the Public Coordination Process from Federal and state regulatory agencies, special interest groups, and the general public.

Goals of conceptual model

The conceptual model has two goals:

a. Site characterization which is a general description of the environmental setting.

b. Defining complete exposure pathways which are the links between sources of contamination and humans or organisms.

Site characterization is an integral part of the ecological and the human health risk assessment. It should:

a. Provide a brief overview of the management area in terms of its current and past uses.

b. Characterize the management area relative to receptors.

c. Describe the presence of contaminants in potential exposure media (sediments, biota, suspended sediments, water).

A complete exposure pathway is a physical, chemical, or biological mechanism or some combination which may transport a contaminant from a source, such as sediment, to a specified human or other organism such as a commercial fish species or an endangered aquatic bird. A complete exposure pathway does not necessarily translate to risk. The conceptual model attempts only to describe the

potential for migration of contaminants based on the site-specific physical conditions, chemistry, and geology. It provides neither a quantitative estimate of the amount of contaminant moving along a specific pathway nor an estimate of resulting concentrations. Subsequent components of the risk assessment will incorporate information on the amount of a contaminant moving along this pathway and evaluate whether that amount poses a potential risk to a human or other organism.

The dredged material manager will recognize that much of the information necessary to meet these goals is available through the Tiered Evaluation Process. Figure 2 shows where information obtained during that process relates to these overall goals. In most cases, attaining these goals does not require new data but is an integration of the comprehensive analysis conducted in Tier I, supplemented with the information collected in Tier II of the testing manuals. The risk assessor should review NEPA documentation and associated information during the development of the conceptual model. Clearly, the various Tier I tasks such as summaries of physical, chemical, and biological information, field monitoring studies, descriptions of the various sources of contaminants to the dredged material, and the review of regulatory permits in the area contribute to the development of the characterization. The conceptual model is a framework for organizing previously acquired information.

Steps in developing a conceptual model

There are seven steps in developing a conceptual model (Figure 3).

1. Describe the dredged material management activity.
2. Identify the kinds and spatial extent of habitats that are present in and around the management area.
3. Identify the species and humans that may use these habitats and that may be potential receptors.
4. Specify the contaminants of concern.
5. Describe mechanisms which may bring a contaminant into contact with a human or other organism.
6. Describe the potential routes of contact between the contaminant and the receptor.
7. Describe the complete exposure pathway.

Step 1: Describe dredged material management activity

The first step in developing the conceptual model is to provide a narrative description of the proposed dredged material management activity. This description should include the manner of sediment dredging and disposal, the amount of material under consideration, and the source of dredged material. The

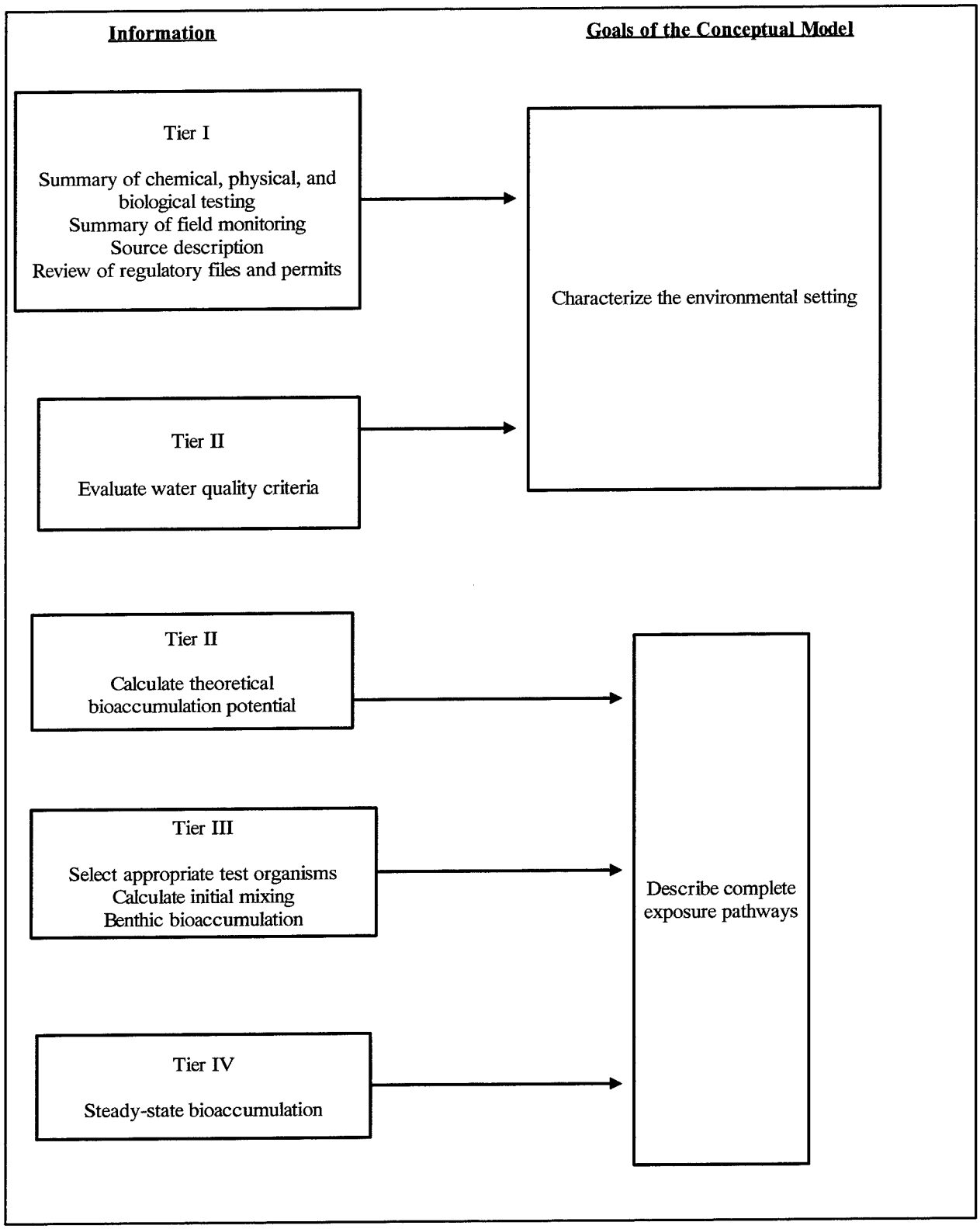

Figure 2. Flowchart depicting relationship between information collected during sediment evaluation process and goals of conceptual model

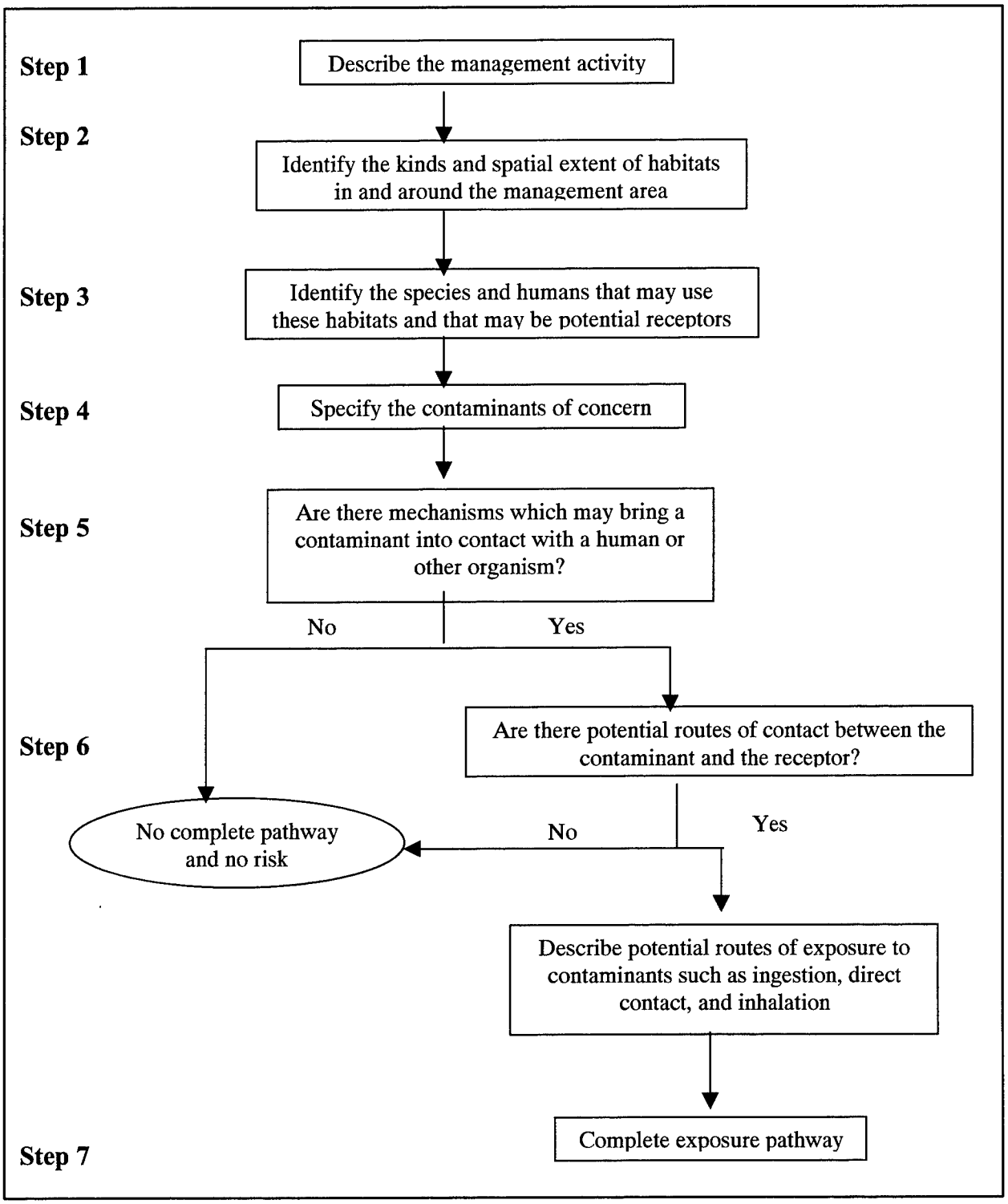

Figure 3. Steps in developing a conceptual model and determining complete exposure pathways

product of this step is a written description of the proposed dredged material management activity.

Example 1: Description of the Dredged Material Management Activity

A local marina has proposed dredging 10 new slips. The existing water depths at the slips is 1.5 m (5 ft) mean lower low water (MLLW). Each slip will be 15 × 6 m (50 × 20 ft) and dredged to a depth of 3 m (10 ft) MLLW with a 0.6-m (2-ft) over-dredge allowance. The project will also require dredging of the channel resulting in an estimated 76,460 cu m (100,000 cu yd) of dredged material. A clamshell dredge will remove the material to a hopper barge for transport to an offshore unconfined management area for which a site designation report is available. The water depth near the site averages 30 m (100 ft), and there is low to moderate wave energy.

Step 2: Identify habitats

It is important to identify habitats in and near the dredged material management area, because these will largely determine human uses and ecological receptors for the conceptual model. The identifications should be specific and conform to common ecological descriptions of aquatic habitat.

There is no restriction or recommendation regarding the number of habitats described in this section. Generally, the habitat classifications should not be so broad as to lose ecological meaning, nor so specific that they lack information regarding the relationships among organisms. Example 2 provides a list of the types of questions to ask during this step.

There are no rules regarding how close to a management area a habitat must be to be included in the site description. It is best to use biological or physical characteristics that impose a functional, as opposed to a geographic relationship between the management area and appropriate habitats to make decisions. Such characteristics might include: depth of vertical mixing, the presence of geological sills, a permanent thermocline, erosional characteristics, water mass mixing, wave action, grain size, flow, presence of a continuous shellfish bed, similarity in vegetative characteristics, etc. The product of this step should be narrative text, maps, and figures, as necessary, which describe the habitats at and adjacent to the disposal site. Much of this information should be available from the site designation process and NEPA documentation.

Example 2: Description of Habitat Surrounding Management Site

The risk assessor used the following questions to guide the description of the habitat at and near the management area (disposal site) where the dredged material from the marina and channel will be transported.

a. What is the size of the management area (disposal site)?
b. What is the size of the local water body?
c. Are there fishery breeding, nursery, or feeding areas near the site?
d. Is the site near or adjacent to seasonal migration pathways for fish, mammals, or piscivorous birds?
e. Are there biological reefs near the site (shellfish reefs, coral reefs) or other particularly productive benthic environments?
f. Is the site near a wetland such as a salt marsh, Typha marsh, tidal flat, or flood plain?
g. Is the site near a productive commercial or recreational fishery?
h. Are there habitats identified by local, state, or Federal agencies for special protection such as critical habitat for endangered species, a national seashore park, or a state wetland refuge near the site?
i. Are there Federal, state, or endangered species near the site?

The management area for this dredging project is in a coastal bay that is approximately 8 × 3 km (5 × 2 miles), and connects to the open ocean through a broad mouth. The management site is 5 km (3 miles) offshore. The nearshore environment includes an extensive salt marsh. The bay has a sand and silt bottom and a stratified, seasonal thermocline. There is a winter flounder fishery near the site. There are migratory species, including winter flounder and mackerel, in the area. There are no endangered species found near the site.

Step 3: Identify species and humans that may use habitats

Identify species. This is the first step in the ultimate selection of receptors for use in the risk assessment. It also provides input to the human health risk assessment in identifying a potential exposure pathway, ingestion of seafood by humans (i.e., by identifying those species used in commercial or recreational fisheries). Again, most of the necessary information should have been collected during the disposal site selection/designation process and assembled in the accompanying NEPA documentation.

First, identify biological communities as general community types such as pelagic, demersal, epibenthic, or infaunal while simultaneously considering the overlap in such distinctions. Secondly, list the types of organisms likely to occur within these general communities. Note that stakeholders may select receptors or resources of lesser ecological importance for economic or aesthetic reasons.

Identify human users. The conceptual model should specify human receptors who may use the management site, local residents living or working near the site, and workers who may contact sediments during dredging, transport, or management of the materials. The potential human receptors include:

a. Potential recreational users of the management site (e.g., swimmers, boaters, fishermen, naturalists, waders).

b. Local residents, especially where upland disposal is under consideration (e.g., off-site resident, trespassers).

c. Workers (barge operators, on-site workers, facility workers, pretreatment workers).

d. Individuals who fish or consume fish or shellfish that may have exposure to contaminants from the dredged material management site.

The product of this step will be a list of animal and/or plant species and humans likely to use the habitats at and within the influence of the disposal site. For the organisms, the list should reflect the variety of trophic levels, feeding types, and phylogenetic diversity in the identified habitats. As much as possible, the list should assign species to various communities and provide their general ecological function within the community. For humans, the list should reflect human receptors who may use or work at the site or ingest seafood from or near the site.

Obviously, the list cannot be inclusive of all species which may use or pass through the disposal site area. However, it should include multiple representative species of most, if not all, the functional types in the area, and it should list any pertinent endangered or threatened species that reside in or pass through the area. The information gathered in this section will be important in the selection of receptor species.

Example 3: Identification of Species and Humans that May Use Habitats

The following table is a summary list of species identified at or near the potential dredged material management site. It characterizes the species by habitat (e.g., planktonic, benthic) and by function within the ecosystem. Most of this information will have been assembled during the site designation process.

Tabulations such as these allow the risk assessor to judge the diversity of habitats among the aquatic community and provide some sense of general diversity and ecological function at the management site. Note that the species in this table, while they occur at or near the site, will not necessarily be selected as receptors for further analysis. For example, at most sites it is unlikely that phytoplankton will receive more than a short-term exposure to the dredged materials (primarily during disposal), because most of the contaminants potentially associated with dredged materials have a high affinity for sediment particles and low solubility.

Species List for Management Area and Adjacent Areas

Receptor	Common Name	Functional Group
Phytoplankton		Primary producer
Asterionella		Primary producer
Melosira		Primary producer
Nitzschia		
Epibenthic Animals		
Homerus americanus	Lobster	Scavenger/predator
Crassostrea virginica	Oyster	filter feeder
Infauna/Benthic Animals		
Mya arenaria	Soft shell clam	Filter feeder
Mercenaria mercenaria	Hard shell clam	Filter feeder
Cardium edule	Cockle	Filter feeder
Gammarus duebeni	Amphipod	Deposit feeder
Nereis virens	Sandworm	Scavenger/predator
Fish		
Anguilla rostrata	Eel	Predatory fish
Scomber scombrus	Mackerel	Migratory pelagic feeder
Pseudoplueronectes americanus	Winter flounder	Bottom feeding fish

In addition to these species, there are also humans who use the area around the site, including workers involved in dredging, transport, or management of the material, fishermen, and boaters. Because there is a winter flounder fishery near the site, other individuals may be exposed through fish consumption.

Step 4: Specify contaminants of concern

This step in the development of the conceptual model is closely tied to the tiered sediment evaluation. Those procedures have explicit methods for identifying COCs and for deciding whether they may present a potential environmental problem. The risk assessment rests heavily upon this prior work and should not introduce COCs previously screened from consideration by the prior evaluation procedures.

The risk assessment should address risk from the COCs identified during the tiered sediment evaluation process. The ocean dumping regulations (40 CFR Ch. 1 [7-1-88 edition] 227.6) and dredged material testing manuals (USEPA/USACE 1991, 1998) provide guidance regarding the selection of contaminants of concern for dredged material.

Figure 4 shows the process for making the selection. It is a step-wise process that uses information from the sediment evaluation procedure to select COCs. This subsection summarizes the Tier I, II, and III sediment evaluation procedures and describes how they apply to the selection of COCs for risk assessment.

Summary of Tier I evaluations. The Tier I procedures identify potential COCs as those constituents which the regulations consider prohibited as other than trace constituents. These include:

a. Organohalogen compounds.

b. Mercury and mercury compounds.

c. Cadmium and cadmium compounds.

d. Oil.

e. Known carcinogens, mutagens, or teratogens.

In addition, the testing manuals describe several bases upon which to identify contaminants of concern. These include:

a. Presence in the dredged material.

b. Presence in the dredged material relative to the concentration in the reference material.

c. Toxicological importance.

d. Persistence in the environment.

e. Propensity to bioaccumulate from sediments.

Simple presence is not sufficient to include a contaminant as a potential contaminant of concern. However, a persistent and toxic chemical would be included. Some contaminants may occur in sediments below their toxic levels, yet

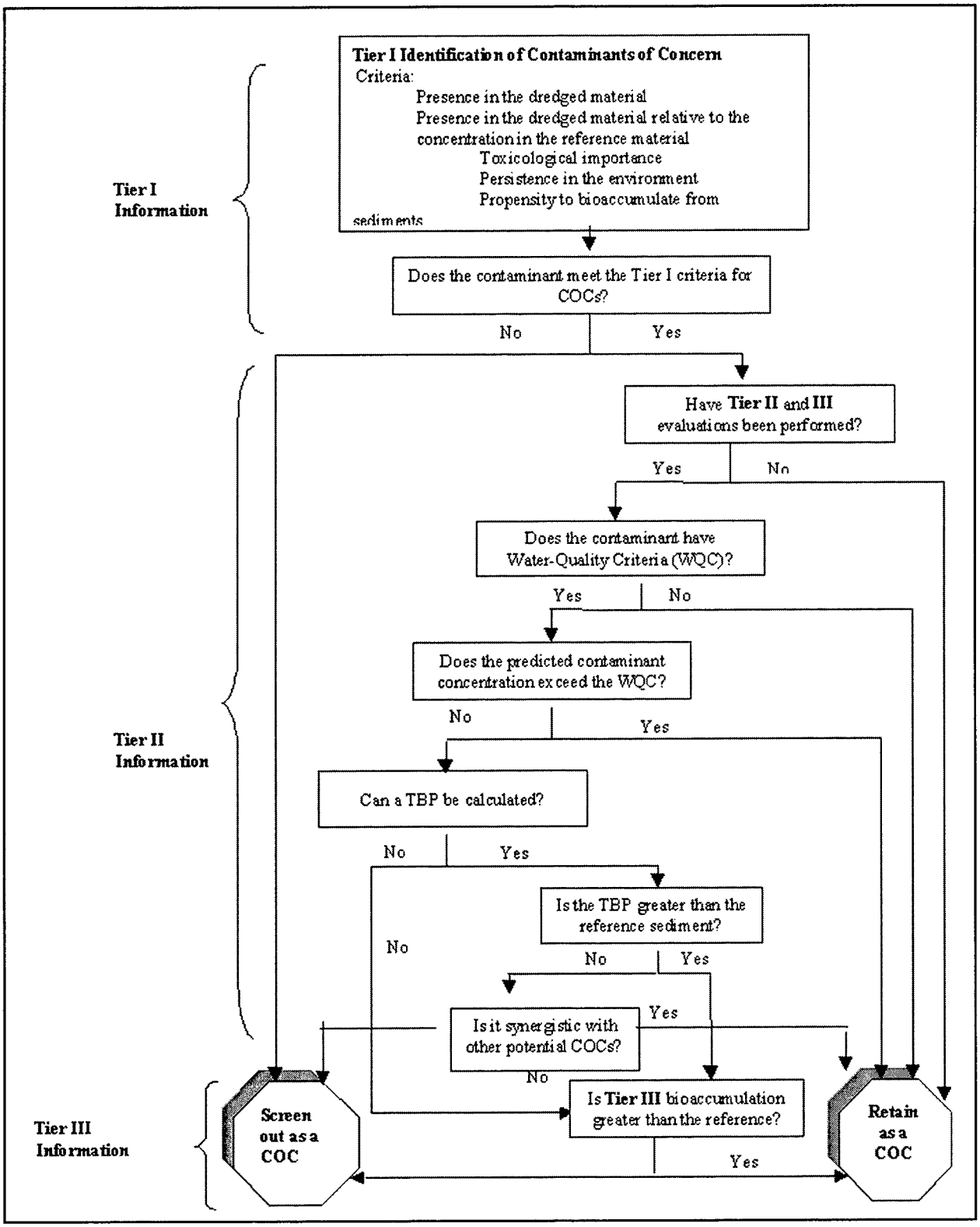

Figure 4. Identification of contaminants of concern

they are so bioaccumulative that they present a potential problem to higher trophic levels. In Tier I, the testing manuals specify four properties which control the propensity to bioaccumulate:

a. Hydrophobicity.

b. Aqueous solubility.

c. Stability.

d. Stereochemistry.

Application of Tier I criteria for selecting COCs. All compounds identified as potential COCs in Tier I will be carried in the risk assessment unless evaluations in subsequent tiers are available to eliminate a compound from the COC list.

Summary of Tier II evaluations. Tier II of the sediment evaluation procedure provides a method to screen sediments for potential impact and thereby eliminate the need for further testing. Tier II evaluates the COCs identified in Tier I for compliance with water-quality criteria (WQC), and calculates Theoretical Bioaccumulation Potential (TBP) to address potential benthic impact.

To evaluate water-column impact, the Tier II evaluation predicts a water-column concentration for all of the contaminants of concern identified in Tier I. This prediction makes the conservative assumption that all of the contaminants in the dredged material are released into the water column. If the predicted concentrations of all potential COCs are below the WQC concentrations, and no synergistic effects are suspected, then the dredged material complies with Tier II WQC requirements. If the predicted concentrations of any of the potential COCs exceed WQC, if there are no criteria available, or if synergistic effects are suspected, further testing is required in Tier III.

To evaluate benthic impact, the TBP calculated for the nonpolar organic COCs in the dredged material are compared to the TBP calculated for the same contaminants in the reference sediment. If the TBP of nonpolar organic compounds for the dredged material exceeds that of the reference sediment, further evaluation of bioaccumulation in Tier III is appropriate. Tier III evaluation is also necessary if the COCs include compounds other than nonpolar organics which may bioaccumulate.

Application of Tier II results for selecting COCs. If the sediment evaluation procedure progressed to Tier II, then compounds which do not have WQC or whose predicted water-column concentration exceeds its WQC should be retained as COCs. Note that the comparison should be made to all available WQC including:

a. Acute criteria for the protection of aquatic life.

b. Chronic criteria for the protection of aquatic life.

c. Criteria for the protection of humans from consumption of organisms only.

d. Criteria for protection of humans from consumption of water and organisms.

Those compounds which meet WQC and are neither bioaccumulatable nor act synergistically with other compounds will be screened out as COCs. The risk assessment can screen out compounds which do or may bioaccumulate if their Tier II analyses of TBP in the dredged sediments is less than the TBP calculated for reference sediments. If the TBP for the dredged sediment is greater than the TBP for reference sediments according to Tier II protocols, then the decision to retain or screen out the COC depends on the results of Tier III testing.

Summary of Tier III evaluations. Tier III assesses the impact of contaminants in the dredged material on appropriate sensitive organisms to determine if there is a potential for the dredged material to have an unacceptable impact. This tier uses water-column and whole sediment toxicity bioassays and bioaccumulation tests.

Water-column toxicity bioassays assess the effects of sediment-associated contaminants on water-column organisms. Water-column toxicity tests must be used when WQC are not available or when synergistic effects are suspected. If the concentrations of dissolved plus suspended contaminants do not exceed 0.01 of the acutely toxic concentrations, the dredged material complies with water-column toxicity criteria. If the concentration exceeds 0.01 of the acutely toxic concentrations, the dredged material does not comply.

Whole sediment bioassays assess the effects of sediment-associated contaminants on benthic organisms. If bioassay organism mortality is statistically greater than in the reference sediment and exceeds mortality in the reference sediment by at least 10 percent (or a value that is in accordance with approved testing methods), the dredged material does not meet the limiting permissible concentration for benthic toxicity.

Tier III benthic bioaccumulation tests determine bioavailability through 28-day exposure tests. Bioaccumulation potential has to be in compliance with regulations before a dredged material can be considered acceptable for ocean dumping. Tier III includes comparing concentrations of COCs in tissues of benthic organisms after a 28-day exposure period to Food and Drug Administration (FDA) Action Levels. It is considered unacceptable if the concentration of contaminants in any test species exceeds FDA action levels.

If tissue contaminant concentrations are less than FDA action levels or if no FDA levels are available, they must be compared to contaminant concentrations in tissues of organisms similarly exposed to reference sediment. If tissue concentrations of COCs in organisms exposed to dredged material do not statistically exceed those of organisms exposed to reference sediment, then the dredged material complies with bioaccumulation regulations. If the concentrations of COCs in organisms exposed to dredged material exceed those of organisms exposed to reference sediment, Tier III provides eight factors to consider to determine compliance.

Application of Tier III results to selection of contaminants of concern. The selection of COCs for the risk assessment uses the Tier III bioaccumulation test results. Any compound in the proposed dredged material tested under Tier III which bioaccumulates in significantly greater amounts than a reference sediment should be retained as a COC. Note that at the end of Tier III, the retained list of compounds will include:

a. Contaminants for which there is no WQC.

b. Contaminants whose predicted concentrations exceed any applicable WQC.

c. Contaminants which bioaccumulate from proposed dredged materials at concentrations significantly greater than a reference area sediments.

The product of this step is a list of contaminants of potential concern which will be used in developing the links between contaminant sources and potential ecological or human receptors in the conceptual model. A narrative which explains the genesis of the list through a consideration of the results of the tiered sediment evaluation procedures should accompany the list.

Example 4: Identifying Contaminants of Concern

For the marina project under consideration, five contaminants found in the dredging material intended for the offshore management site met the criteria for Tier I identification of COCs. Specifically, cadmium, lead, mercury, endosulfan, and PCBs are potential contaminants of concern because they are present in the material and have known toxicological effects.

The tabulation below provides the WQC and the predicted concentrations for the potential COCs from Tier II evaluations. The evaluation revealed that neither lead nor cadmium have WQC for the protection of humans from consumption of organisms. These two contaminants must, therefore, be retained as COCs.

The remaining contaminants, mercury, endosulfan, and PCBs, have all WQC including: acute criteria for the protection of aquatic life; chronic criteria for the protection of aquatic life; criteria for the protection of humans from organisms only; and criteria for protection of humans from water and organisms. Among these three potential contaminants, the predicted water concentration of total PCBs from the dredged material exceeded the criteria. Therefore, total PCBs were retained as a COC.

A theoretical bioaccumulation potential could not be calculated for mercury because it is an inorganic compound. Therefore, a Tier III evaluation was necessary to determine compliance. The Tier III evaluation revealed that bioaccumulation of mercury in the dredged material was less than that of the reference sediment, and it was screened out as a COC.

Because endosulfan is a nonpolar organic compound, a TBP could be calculated, but the TBP, in this case, did not exceed that of the reference sediment. In addition, no synergism with other potential COCs was suspected, and endosulfan was screened out as a COC.

At the end of the three tiered evaluation, three contaminants in the dredged material, cadmium, lead, and PCBs, were selected as contaminants of concern for the risk assessment. This continuous example will carry total PCBs through the risk assessment.

Contaminant	Saltwater Criterion Acute Conc. (ug/L)	Saltwater Criterion Chronic Conc. (ug/L)	Criteria for Human Water and Organisms	Protection of Health Organisms Only	Predicted Contaminant Concentration	COCs Retained
Cadmium	43	9.3	10	NA	10.4	X
Endosulfan	0.034	0.0087	74	159	0.0067	
Lead	220	8.5	50	NA	14.7	X
Mercury	2.1	0.025	0.146	0.14	0.019	
PCBs	10	0.03	7.90E-05	7.90E-05	1.2	X

NA = Not available

Reference:

USEPA (1999). National recommended water quality criteria. USEPA, Office of Water, Washington, DC. EPA/822-Z-99-001.

Step 5: Describe release mechanisms

This step will describe mechanisms which may release contaminants from the dredged material management area and allow them to contact ecological or human receptors. Such mechanisms may include disturbance of the sediment, bioturbation, dissolution, resuspension, diffusion through engineered barriers, or advection. It is important to remember that the mechanisms are considered only if they result in a release which brings contaminants into contact with potential receptors. The product of this step is a narrative which describes potential release mechanisms associated with the management option under consideration.

Example 5: Description of Potential Release Mechanisms

During this dredged material management operation, there are several potential release mechanisms which could result in exposure to COCs. Once the material has reached the management area, sediment can become suspended in the water during placement. The area is a low-to-moderate energy environment, has a seasonal thermocline (indicating little surface-to-bottom mixing during summer), and is generally depositional. There is some potential for resuspension of the sediments and advection through wave or storm action and during winter with the breakdown of the seasonal thermocline. There is also potential for diffusion from pore water and advection offsite. These mechanisms could bring the potential COCs into contact with receptors.

Step 6: Describe potential routes of exposure

The simple existence of a release mechanism which may transport a contaminant to a receptor will not result in a complete exposure pathway unless there is some route by which the receptor contacts the contaminant. These routes may include dermal contact, ingestion, absorption across the gills, or inhalation. The conceptual model should specify the likely route or routes of exposure for each receptor separately.

Step 7: Describe complete exposure routes

The last step is to decide whether there is a complete exposure pathway between a contaminant and a receptor. The conceptual model should describe each complete pathway in detail including the source of the contaminant, the release mechanism, the route of exposures and the potential receptors. A complete exposure pathway is a combination of physical, chemical, or biological mechanisms which may transport a contaminant from a source, such as sediment, to an ecological receptor, such as a commercial fish species or an endangered aquatic bird, or to a human receptor, such as a recreational fisherman or someone consuming commercial fish, from an area under the influence of a dredged material management activity.

Whether a pathway is complete depends on:

a. The presence of a particular receptor.

b. The physical accessibility of the contaminants to a receptor.

c. The chemical properties of a COC (e.g., solubility, partitioning coefficients) which govern its partitioning among media and from physical media to biota.

d. The physical attributes of a site which may govern movement of a contaminant (e.g., advection, upwelling, sediment transport).

The risk assessor must consider these factors in deciding whether there is a complete pathway at a specific site. When an exposure pathway is complete, the risk assessor must decide whether there is potential for risk associated with that pathway. A complete exposure pathway does not necessarily translate to risk. Risk depends on the concentration or dose to the receptor relative to that receptor's toxic response. Later sections of the risk assessment will address the dose or concentration to which a receptor is exposed and will address the toxicity of the chemical.

At most dredged aquatic material management sites, the potential links between contaminants and potential ecological receptors are:

a. Sediment to benthic organisms.

b. Benthic organisms to pelagic or demersal organisms.

c. Water column to pelagic organisms.

Figure 5 shows a generalized conceptual model with the most likely complete exposure pathways at dredged material management sites. Note that direct exposure from sediments to pelagic organisms is possible (e.g., exposure to

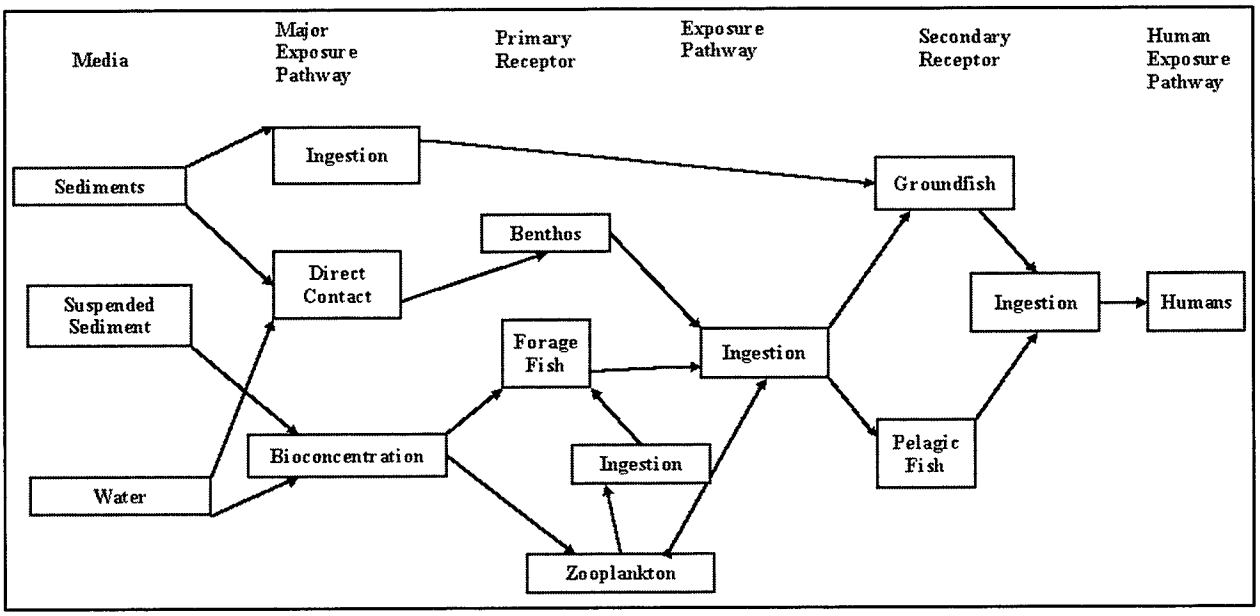

Figure 5. Example of conceptual model for ecological exposure pathways

resuspended sediments in energetic environments). In shallow waters, there may be exposure via plant uptake and subsequent herbivores. In the figure, the terms "primary receptor" and "secondary receptor" represent general trophic levels, not a prioritization of importance. Note that this conceptual model depicts a shallow site where forage fish and zooplankton are important receptors and are important biological media for exposure to higher trophic levels (groundfish and pelagic fish).

Contaminant exposure pathways that do not lead to a species or group of species or humans which may be potential receptors are incomplete and therefore the risk assessor may assume that there is no potential for risk associated with a particular contaminant along that pathway (Figure 3).

The product of Steps 6 and 7 is a graphical and narrative description of the complete exposure routes specific for the COCs, habitats, types of species, and likely human receptors. It should include a written summary of the chemical, physical, and biological conditions at the proposed disposal site. Where data are insufficient to conduct any of the preceding steps, the description should recommend means (e.g., field surveys) to provide the information necessary to complete the conceptual model. In those cases where further field or laboratory work is recommended, the description should also stipulate the required data goals and methodology. Subsequent steps in the ecological risk assessment, particularly the development of a list of receptors, will depend on the site characterization inherent in the development of the conceptual model.

Example 6: Description of Complete Exposure Pathways

The risk assessor used the following questions to guide the determination of complete exposure pathways between the proposed dredged material and the potential receptors:

a. Could contaminants reach receptors via direct contact?
b. Are one or more receptors inhabiting or using an area where contamination exists or will exist?
c. Is the location of contamination such that one or more receptors could contact it currently or in the future?
d. Are there advective or dispersive processes which may deliver the contaminant to a receptor or habitat?
e. Could contaminants reach receptors via indirect contact?
f. Is contamination bioaccumulative or bioconcentratable?
g. Are there higher order predators which may accumulate the contaminant?
h. Could contaminants reach receptors or habitats via groundwater?
i. Can contaminants leach into groundwater?
j. Does groundwater discharge to aquatic habitats?
k. Are contaminants present at surface sediments?
l. Can contaminants be leached or eroded from surface sediments or soil?

The answers to these questions indicate that there is a benthic community with potential for direct contact and ingestion of sediments by invertebrate organisms at the management area. There is then potential for bioaccumulation to higher-order predators through ingestion of the benthic organisms. There is some potential for bioconcentration of COCs from suspended sediments in the water column to forage fish and zooplankton, given the moderate vertical mixing which may occur at the site in winter. The management option does not have an effluent discharge, so there is minimal likelihood of dissolved contamination in the water column (there is a potential for exposure in the water column during disposal, but it is of short duration). There is a commercial fishery, winter flounder, which results in a complete pathway to humans through ingestion of flounder. The management area is too far offshore (5 km (3 miles)) to consider groundwater discharge as a likely exposure pathway. Also, the management option does not result in sediment exposures at the water surface as might be the case for an offshore containment island.

Sources of information for developing conceptual model

Each risk assessment will require site-specific information. The following sources provide data on various estuaries, coastal areas, and long-term monitoring programs for biological, chemical, and physical characteristics of an area:

a. Environmental impact statements for disposal site designations.

b. Previous assessments of dredged material disposed at the site.

c. NOAA Programs:

(1) Historic Trends Reports for Various Estuaries, National Ocean Pollution Program - these are reports on individual estuaries and coastal areas prepared by the National Ocean Service (NOS) and National Sea Grant College Program.

(2) National Status and Trends Program Benthic Surveillance Project, NOS - reports on contaminant levels in benthic organisms in marine coastal areas.

(3) National Status and Trends Program Mussel Watch Project, NOS - reports on contaminant levels in mussels and oysters in coastal areas.

(4) NOAA Technical Memorandum Series Published by NOS - various reports and data summaries of biology, chemistry, and physical oceanography for coastal areas.

(5) National Marine Fisheries Service (NMFS) Reports – statistical and catch reports prepared by NMFS.

d. USEPA environmental monitoring and assessment program reports.

e. State Division of Marine Fisheries Fishery statistic reports and monitoring reports.

f. State Fish and Game Reports.

g. Clean Water Act Section 208 Reports.

h. National Heritage Program Atlases.

i. Soil Conservation Service Reports.

j. United States Geological Survey Reports.

k. State and local Conservation Agency Reports.

l. U.S. Fish and Wildlife Service Reports.

Selecting and Characterizing Representative Receptors

What is a representative receptor?

Representative human receptors are humans who have a complete exposure pathway as described in the conceptual model and whose exposure is likely to represent a reasonable worst-case exposure to the COCs.

Representative ecological receptors are organisms whose life histories and habitat requirements fairly represent the range of habitats and life histories for those organisms with complete exposure pathways which are found near the dredged material management site.

Why does risk assessment use representative receptors?

It is practically impossible for the risk assessment to address risk to every possible receptor. There will be a wide variety of species and types of species under the potential influence of the dredged material management site. Therefore the ecological risk assessment must have some method to choose one or more receptors which best represent the types of species likely to contact COCs from the dredged material management area.

Similarly, human contact with contaminants may vary over a wide range, so it is important to choose a human receptor which represents a realistic but likely worst case from among the range of possible human receptors.

How will the risk assessment use representative receptors?

The risk assessment will use the biological properties and activity patterns of representative receptors to develop estimates of how much contaminant the receptor may encounter. It will use toxicological information about the receptor to estimate whether that level of contaminant exposure might present a risk to the representative receptor. By broad extension, the assessment will assume that risk to the representative receptors implies risk to ecological populations or individual humans.

Select and Characterize Representative Receptors

It is unreasonable to assume that a risk assessment can address potential risk to every species or every human activity which may be associated with the dredged material management activity. Therefore, the risk assessment uses representative receptors. Representative human receptors are humans who have a complete exposure pathway as described in the conceptual model, and whose exposure is likely to represent a reasonable worst-case exposure to the COCs. Representative ecological receptors are organisms whose life histories and habitat requirements fairly represent the range of habitats and life histories found near the dredged

material management site. Using a representative species approach is a commonly accepted technique in regulatory practice. For example, this approach has historically been used in other Clean Water Act regulatory activities such as 301h and 301b demonstrations.

Select and characterize human receptors

The assessment should specify the human receptors who may use the management site, local residents living or working near the site, and workers who may contact sediments during dredging, transport, or management of the materials. Obviously, the types of human receptors will vary with the technology employed in the dredged material management activity and the location of the activity. The likely list of potential human receptors include:

a. Potential recreational users of the site (swimmers, boaters, fishermen, naturalists, waders).

b. Local residents (off-site resident, trespasser - depends on proximity of management site to shore).

c. Workers (barge operators, onsite workers, facility workers, pretreatment workers - depends on the technology used).

Select ecological receptors

This step identifies the receptor species and provides the rationale for their selection as representative receptors from among the species likely to occur in the disposal site area.

The actual receptors chosen will vary among disposal sites. However, general guidance for receptor selection is to select those species which:

a. Are likely to occur at the site.

b. Represent a reasonable (although not comprehensive) cross section of the major functional and structural components of the ecosystem under study.

c. Represent various trophic levels (e.g. saprophytes, herbivores, primary and secondary carnivores), feeding types (detritivores, scavengers, filter feeders, active predators, forage fish, piscivorous birds), and habitats (benthic, demersal, pelagic) so that exposure pathways can be evaluated.

d. Represent those types of organisms most likely to encounter the contaminants of concern.

e. Are relatively abundant and ecologically important within the selected habitats.

f. Have available applicable toxicological literature.

g. Are relatively sensitive to the contaminants of concern.

h. Represent various mobility and local feeding ranges.

i. Bioaccumulate contaminants of concern.

j. Are economically important or have Federal/state endangerment status.

k. Exhibit any observed visible evidence of stress.

Much of this information will already be available from the site selection process.

The risk assessment will use the biological and ecological characteristics of the selected species in the later tasks of estimating exposure and risk to the ecosystem.

The product of this step is a list of human and ecological receptor species aggregated by functional group. This will be used to develop an estimate of exposure to COCs, estimate bioaccumulation, and characterize risk. The species chosen should represent the ecological community and its sensitivity to the contaminants of concern.

Example 7: Selecting Human and Ecological Receptors

Ecological receptors

The potential receptors in the management site include the invertebrate community that lives on or in the sediments (the benthos), fish species that inhabit the bay for part of their life cycle or as a foraging area, and the plankton community of invertebrates, fish larvae, and algae that are suspended in the water column and carried with the tidal currents into and out of the bay.

Based on the data available for the site, it is clear that the focus of the analysis should be on animals that have direct contact with the sediments. These animal communities (both invertebrate and fish) tend to reside longer in particular areas than do plankton (carried with the currents) or fish that inhabit the water column (e.g., blue fish). Specifically, the environmental receptors which are emphasized in this analysis are the benthic invertebrate community and the demersal (bottom) fish community.

Within the demersal fish community, this risk assessment uses the winter flounder, (Pseudopleuronectes americanus) as the representative species because it is the most commonly occurring species in the area, supports a major commercial fishery in the bay, and is a major predator on bottom dwelling organisms.

Human receptors

The likely human receptors include consumers of winter flounder from the commercial and recreational fishery.

Characterize ecological receptors

For each chosen receptor, the assessment should include a species profile which characterizes the biological properties of the selected receptors. These profiles consist of text descriptions of the relevant ecological and physiological characteristics and taxonomic relationships of the receptors. These include, but are not limited to, descriptions of: trophic status, feeding type, food preferences, ingestion rates, range, prey, predators, migratory habits, breeding habits, likely habitats, population estimates, reproductive strategies, substrate and habitat preferences, and life history. The profiles should also include any particular vulnerabilities or status of the species as rare, threatened, or endangered. Note that profiles should include, as much as possible, site specific aspects of an organisms biology. For example, it is important to know whether a receptor organism breeds near the site.

The product of this step is a written characterization of ecological receptors derived from: a literature review, reviews of existing studies, and results of surveys during the site selection process or monitoring at existing sites. This characterization will be used in the development of exposure scenarios and the risk characterization.

Example 8: Characterization of Ecological Receptors - Winter Flounder

The winter flounder is a coastal demersal species with a primary range in cold-temperate boreal waters. Winter flounder occur at depths from the intertidal to 150 m and on hard or soft mud, clay, sand, or pebble bottoms of bays, estuaries, and coastal waters (Bigelow and Schroeder 1953). Perlmutter (1947) suggested the existence of many discrete local stocks based on several key observations: demersal eggs, nondispersive larvae, juvenile phases, and complete lack of adult mixing with other stocks.

Winter flounder spawn in most estuaries from Chesapeake Bay through the Gulf of Maine from midwinter to early spring (Azarovitz 1982). It is believed that winter flounder return to the same spawning location year after year (NMFS 1986). Winter flounder eggs are demersal and adhesive, and therefore the spawning and nursery areas for the species should coincide.

In areas north of Cape Cod, winter flounder remain in bays and harbors year-round, moving into deeper holes and channels during the warmest weather (Azarovitz 1982).

Winter flounder feed by sight near the bottom. For example, Pearcy (1962) showed that fish fed in a dark room did not eat until zooplankton died and sank to the bottom. Field observations confirmed that feeding occurs during the day. These organisms are clearly bottom dwellers who spend significant portions of their lives in close contact with sediments.

It is also significant that winter flounder eat bottom-dwelling organisms because the consumption of these organisms provides another potential exposure pathway. Several investigators (Pearcy 1962; MacPhee 1969; Frame 1972) noted that they are omnivorous, opportunistic feeders, and prey upon polychaete worms, amphipod and isopod crustaceans, pelecypods, and plant material.

Note that this example continues with assessing risk to winter flounder. The risk assessment should similarly address other selected receptors such as a representative benthic organism(e.g., softshell clams) or water-column organisms which may concentrate COCs from suspended sediments.

Assessment and Measurement End Points

What are Assessment and Measurement End Points?

An assessment end point is an explicit expression of the actual environmental value to be protected (USEPA 1992a) during the management of the dredged materials. The term applies only to ecological risk assessment. The environmental values most commonly refer to valuable ecological resources that:

a. Are critical to the normal functioning of an ecosystem such as a diverse benthic community structure.

b. Provide critical resources such as a fishery or sensitive habitat.

c. Are perceived as valuable by humans such as endangered species.

Sometimes the assessment end point cannot be directly measured. In such cases, the risk assessment uses a measurement end point which is a measurable biological response to a contaminant that can be used to make inferences about the assessment end point. For example, an assessment end point might be sustaining fishery diversity and abundance while its related measurement end point is a measure of the community structure of the fish populations near a dredged material management site.

How Are Assessment and Measurement End Points Used in Ecological Risk Assessment?

The ecological risk assessment uses the assessment end points and measurement end points to decide whether there is risk due to a specific dredged material management activity based on whether the activity will alter the assessment or measurement end point beyond some acceptable limit.

What Are Some Common Assessment and Measurement End Points?

Some commonly used assessment end points include: Sustained aquatic community structure, including species composition and relative abundance and trophic structure; sufficient rates of survival, growth, and reproduction to sustain populations of carnivores typical for an area; sustained fishery diversity and abundance.

Some common measurement end points include: Community analyses of benthic invertebrates; body burdens of contaminants associated with a particular effect; sediment concentrations with a known effect; and the results of a toxicity test.

Select and Evaluate Assessment and Measurement End Points

An assessment end point is an explicit expression of the actual environmental value to be protected (USEPA 1992a) during the management of the dredged materials. The term applies only to ecological risk assessment. The environmental values most commonly refer to valuable ecological resources that:

a. Are critical to the normal functioning of an ecosystem such as a diverse benthic community structure.

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measure of the community structure of the fish populations near a dredged material management site.

The selection of assessment and measurement end points should be an inclusive process which includes input from those groups which may be affected by dredged material management decisions. The process of selecting assessment end points began with the conceptual model when habitats and other receptors at or near the site were identified. The problem formulation continues to refine and explicitly state the assessment end points. They can be specific to the receptors that are present at and adjacent to the site.

The number of assessment endpoints selected at a site will vary depending on site characteristics, the habitats and receptors, and concerns of site managers and other interested parties. Additional guidance on the selection of assessment endpoints is available in USEPA/ERT (1997) and in guidance developed by various USEPA regions and states including California, Massachusetts, and Texas.

Selecting Assessment End Points

This subsection identifies the criteria used to select and evaluate, in narrative form, assessment end points. Figure 6 summarizes the selection criteria. USEPA Guidance (USEPA 1992a and references cited therein) suggests six criteria for such evaluations.

a. Ecological relevance.

b. economic importance.

c. Measurable

d. Susceptible and sensitive to chemically induced stress or other stresses.

e. Unambiguously defined.

f. Logically and practically related to the management decision.

The risk assessment should include a narrative evaluation of whether and how each of these criteria are met.

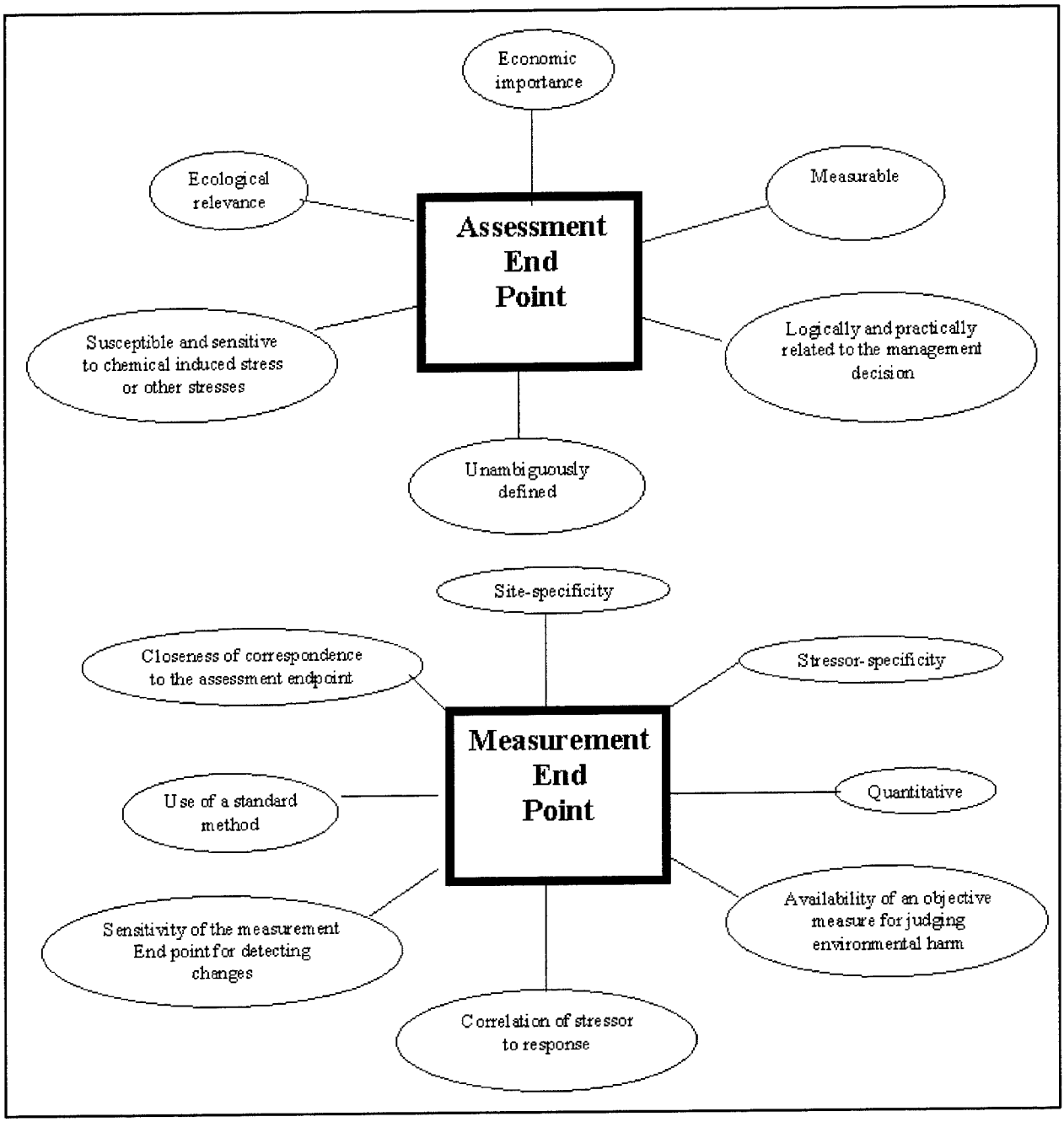

Figure 6. Criteria to select and evaluate assessment and measurement end points

Selection of Measurement End Points

This subsection defines and provides seven attributes which reflect USEPA recommended considerations for the selection of measurement end points. These are:

a. *Closeness of correspondence to the assessment end point*: This attribute refers to the extent to which the measurement end point is representative of, correlated with, or applicable to the assessment end point. If there is no association between a measurement end point (e.g., a study that may have been performed for some other purpose) and the assessment end point of interest, then that study should not be used to evaluate the stated assessment end point.

b. *Site specificity*: This attribute relates to the extent to which data, media, species, environmental conditions, and habitat types used in the study design reflect the site of interest.

c. *Stressor specificity*: This attribute relates to the degree to which the measurement end point is associated with the specific stressor(s) of concern. (Stressors might include a particular chemical, waste, or physical alterations.) Some measurement end points may respond to a broad range of stressors so that it is difficult to interpret results with regard to the stressor of concern, while other measurement end points are more specific to a particular stressor.

d. Availability of an objective measure for judging environmental harm: This attribute relates to the ability to judge results of the study against well-accepted standards, criteria, or objective measures. Examples of objective standards or measures for judgment might include ambient WQC, sediment quality guidelines, biological indices, and toxicity or exposure thresholds recognized by the scientific or regulatory community as measures of environmental harm.

e. Sensitivity of the measurement end point for detecting changes: This attribute relates to the ability to detect a response in the measurement end point. The sensitivity of the measurement end point may be affected by natural or analytical variability.

f. Quantitative: The attribute relates to the degree to which numbers can be used to describe the magnitude of response of the measurement end point to the stressor. Some measurement end points may yield qualitative or hierarchical results, while others may be more quantitative.

g. Correlation of stressor to response: This attribute relates to the degree to which a correlation is observed between levels of exposure to a stressor and levels of response, and the strength of that correlation.

h. Use of a standard method: The extent to which the study follows specific protocols recommended by a recognized scientific authority for conducting the method correctly. Examples of standard methods are study designs or chemical measures published in the Federal Register or the Code of Federal Regulations, developed by the American Society for Testing and Materials (ASTM), or repeatedly published in the peer-reviewed scientific literature.

The products of this subsection constitute a narrative or tabular presentation of assessment and measurement end points with a clear explanation of whether the assessment end points meet the criteria for selection and a qualitative evaluation of whether the measurement end points meet each of the attributes. This will help develop an assessment of the uncertainty associated with each measurement end point.

Example 9: Evaluating The Assessment End Point, Health, and Maintenance of Local Flounder Populations

Consultation with the State Division of Marine Fisheries and the Save The Embayment Association (a citizen's action group) indicates that the area around the planned dredged material management site is a commercial flounder fishery. These groups are concerned that the disposal of dredged sediments from the marina slips may adversely affect flounder populations.

The assessment end point "health and maintenance of local flounder populations" is a reasonable assessment end point and it meets the evaluation criteria.

a. Ecological relevance - Flounder are major bottom feeders in this section of the Bay. Flounder populations generally play a major role in such marine ecosystem level properties as maintenance of invertebrate diversity and nutrient cycling.

b. Economic importance - Flounder are important economically in this portion of the bay. They constitute a commercial fishery year round and an important recreational fishery during summer in nearshore waters.

c. Measurable - The health and maintenance of local fish populations are measurable quantities.

d. Susceptible and sensitive to chemical induced stresses - There are toxicological and field studies supporting the sensitivity of fish to chemically induced stress.

e. Unambiguously defined - The health and maintenance of local fish populations is clearly distinct from assessment of migrating fish or wide ranging fish. The term "local" means populations whose feeding and migrating range is generally on the same scale as the area of the continental shelf proximate to the dredged material management site.

f. Logically and practically related to the management decision - Flounder live and feed near or on the sediments and are continuously exposed to surface water. Their protection as a local resource will be affected by management decisions regarding dredged material disposal in this region of the shelf.

Example 10: Establishing an Appropriate and Relevant Measurement End Point

For PCBs, body burdens in flounder are a reasonable measurement end point. The flounder feed directly on benthic, sediment dwelling organisms which can bioaccumulate PCBs. Note that for other COCs this may not be a good end point. For example, the COCs, also include lead which does not biomagnify.

Attribute	Flounder Body Burdens of PCBs
Closeness of correspondence to the assessment endpoint	Moderate - the measurement of body burdens is not a direct measure of fish health or reproductive capacity.
Site specificity	Strong - the fish probably acquire body burdens due to exposure to site-related contaminants.
Correlation of stressor to response	Moderate - there is evidence in the literature indicating relationships between body burdens of COCs and changes in fish physiology, reproduction, and growth.
Availability of an objective measure for judging environmental harm	Moderate - there are no promulgated standards for protection of ecological receptors based on body burdens. However, the USACE assembled a "residue effects" data based for various contaminants.
Sensitivity of the measurement end point for detecting changes	Moderate - the literature indicates a wide range in tolerance among fish species for body burdens of various COCs.
Quantitative	Strong - the measurement is quantitative.
Use of a standard method	Strong - there are accepted methods for analysis of COCs in tissue.

Ecological Exposure Assessment

What is an Ecological Exposure Assessment?

An ecological exposure assessment builds upon the qualitative descriptions in the conceptual model to calculate a quantitative estimate of the exposure of selected receptors to the contaminants of concern. This quantitative estimate may be a:

a. Concentration in some environmental media such as sediment or water.

b. Tissue concentration in the receptor.

c. Dose of a contaminant of concern to a receptor.

What Are the Steps in Conducting an Ecological Exposure Assessment?

The ecological exposure assessment includes estimating the:

a. Representative concentrations of contaminants of concern (e.g., average, maximum, 95th percentile) in the proposed dredged material.

b. Concentrations of the contaminants of concern in environmental media to which the selected receptors may be exposed along the completed pathways.

c. Amount of a contaminant of concern which a receptor may ingest, contact, or concentrate in its body.

How Does the Exposure Assessment Relate to Ecological Risk?

The exposure assessment should quantify the exposure in the same terms as any available toxicological information. This allows the risk assessor to compare the exposure level to a level which corresponds to a known adverse effect for that receptor. If the calculated exposure level is greater than the level associated with an environmental effect, there is potential for ecological risk from the dredged material.

3 Ecological Exposure Assessment, Effects Assessment, and Risk Characterization

Exposure Assessment

An ecological exposure assessment builds upon the qualitative descriptions in the conceptual model to calculate a quantitative estimate of the exposure of selected receptors to the COC. As described in Chapter 2, the selection of COCs depends on information from the sediment evaluation procedures, and the conceptual model identifies the potential exposure pathways. The goals of the exposure assessment are to:

a. Calculate the physical movement of the contaminants of concern from the disposal site to the point where they may come into contact with a receptor.

b. Provide a concentration of the contaminant of concern at that point.

c. Estimate how much of the contaminant may be ingested or otherwise absorbed into the body of the receptor.

The ecological exposure has three general steps (Figure 7):

Step 1: Estimating the concentration of COCs in the dredged material

This step attempts to provide a conservative estimate of the initial concentration to use in any further calculations or modeling of contaminant movement or transfer through a food chain. This calculation begins with an estimate of the concentration of the contaminant at the disposal site.

The assessment should use the upper 95th-percent confidence limit on the arithmetic mean of the concentration of each COC to represent the projected concentration at the disposal site based on its EPA guidance. Where the data set is

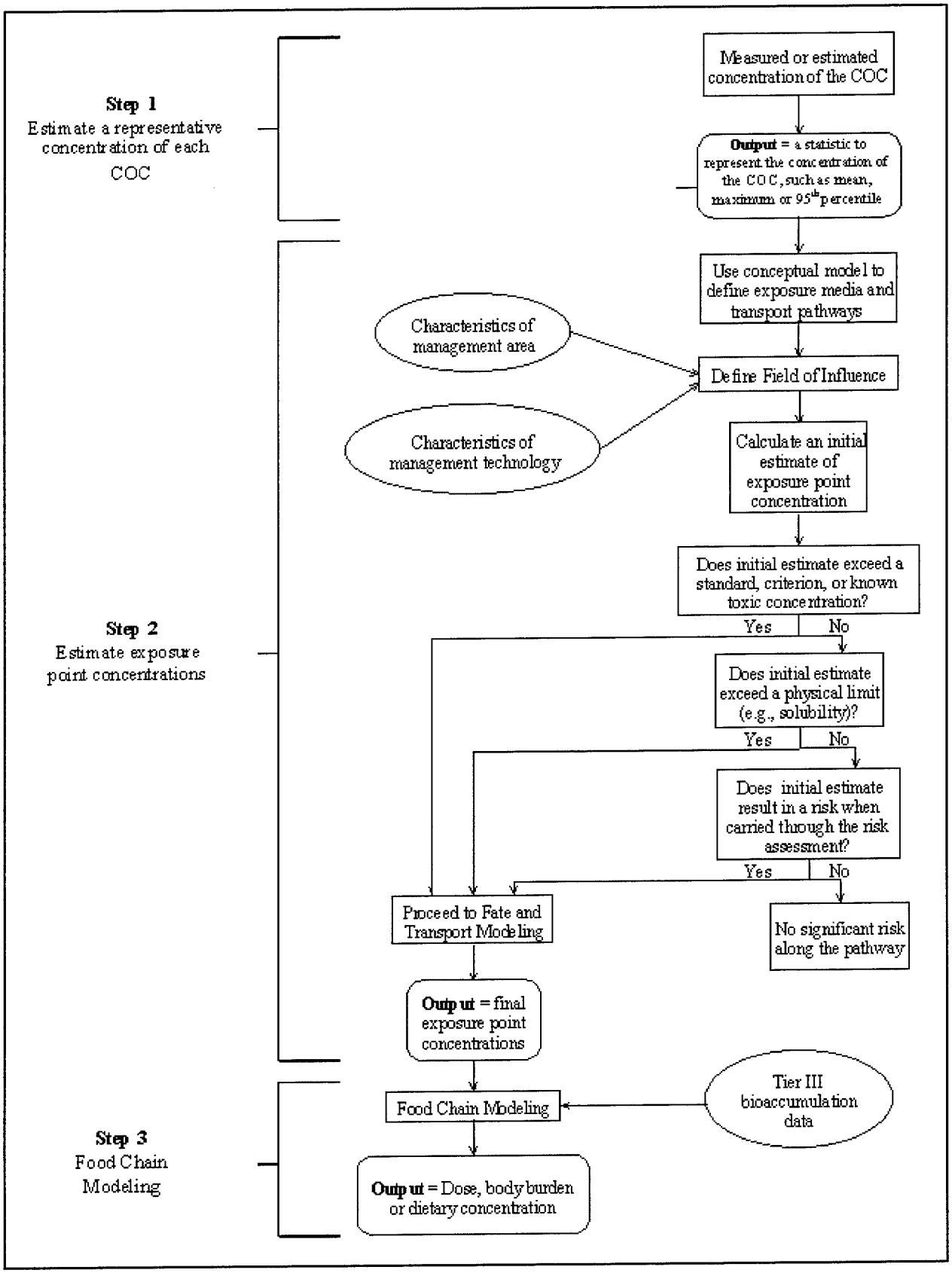

Figure 7. Steps in the development of an exposure assessment

insufficient to provide a reasonable estimate of the 95th-percent confidence limit on the arithmetic mean, use the maximum measured value.

In reality, the distribution may be more heterogeneous than the data imply. Clearly, this assumption ignores mechanisms such as dilution with ambient sediments, bioturbation, mounding, and spreading which may lower the actual concentration to below the average in the dredged material at some points within the disposal site. For example, mounding in the center of the site may put most of the mass of sediments out of the biologically active surficial layer. In the apron of the mound, bioturbation and physical mixing with existing sediments may lower average exposure concentrations.

USEPA guidance requires using the upper 95th-percent confidence limit on the arithmetic mean concentrations (USEPA/Office of Emergency and Remedial Response (OERR) 1992a,b). The use of other statistics, such as the average concentration or the maximum concentrations of the compounds in sediment, can demonstrate the effect of various assumptions on the exposure conditions.

Step 2: Estimating exposure point concentrations (EPC)

Exposure point concentrations are estimates of the concentrations of the contaminants of concern in environmental media to which the selected receptors may be exposed along the completed pathways. The media may include sediments, suspended sediments, water, or concentrations in food. The degree of sophistication needed to make the estimates will vary with the complexity of the environment, the level of information available concerning the site, and the initial estimates of fate and transport. The risk assessment should approach the estimate of exposure point concentrations in two stages:

a. If an initial "back-of-the-envelope" conservatively structured estimate indicates little potential for ecological risk, then the assessment will use this initial estimate.

b. If these initial estimates indicate that transport might be significant enough to result in concentrations associated with potential ecological risk or if the initial estimate exceeds physical limits (e.g., solubility), then the risk assessment should employ more sophisticated models which provide a more realistic prediction of exposure point concentrations.

Making initial estimates of exposure point concentrations

Sediment exposures. For most dredged material management projects, the most likely exposure medium will be sediment. For sediment exposures, the simplest, and most conservative initial calculation, is to assume that the concentrations in the field of influence will equal the concentrations at the management site (the field of influence is that area around the management site which is not subject to direct disposal of sediments, but may experience increased concentrations due to local physical transport mechanisms acting during and after disposal). Alternatively, the initial calculation may make some conservative assumptions about transport of sediments from the management area and subsequent steady-state

dilution and settling within the field of influence to provide a concentration of COCs in sediment. This calculation will require information about sediment resuspension, local currents, and particle settling. The risk assessment should describe the sources of such information or justify any assumptions made about these parameters. It should also explicitly acknowledge uncertainty associated with the parameters.

The important question is "how does the risk assessor define field of influence?" Obviously, the answer to this question lies in the site-specific characteristics of the management area and the management technology employed. The risk assessor may have to employ physical transport models ranging from simple dilution calculations to more complex models which address multiple physical/chemical mechanisms such as dilution, partitioning, sedimentation, advection, and diffusion. For example:

a. If the management area is in a low-energy, depositional backwater environment, the field of influence may be conservatively defined as the extent of the backwater.

b. In an estuarine environment subject to tidal transport, the tidal excursion lengths may dictate the field of influence.

c. If the management area is in a high-energy dispersive environment, the risk assessor probably should not assume that the field of influence concentrations are equal to the concentrations in the management area because there will be significant physical processes affecting the fate of contaminants.

These examples obviously do not encompass all possibilities. The risk assessor will need detailed knowledge of the physical characteristics of the management site and the surrounding areas to make a reasonable conservative estimate of far field.

Water-column exposures. At most dredged material management sites, water-column exposures will be less likely as significant sources of risk than sediment exposures. The likelihood of a water-column exposure depends on the management technology used. For unconfined options or capped management areas, fairly simple estimates of diffusion or pore water transport to the overlying water column along with estimates of advection and dilution can provide estimates of water-column exposure concentrations. In these examples, this transport is likely to be very small. However, for those management options such as dredged material islands or nearshore confined aquatic disposal, which employ dewatering, the estimates of water-column exposures will require an initial estimate of concentrations of COCs in effluent, and may require more sophisticated fate and transport modeling (see text entitled "Modeling exposure

The product of this text is an initial estimate of the concentration of the COCs at the disposal site and its field of influence. The simplest (and most conservative) estimate is to assume the concentrations are equal in these areas.

Example 11: Initial Estimate of Exposure Point Concentration for Total PCBs

The risk assessor has calculated the upper 95th-percent confidence limit on the arithmetic mean concentration of total PCBs based on Tier I measurements. This value is 1 ug total PCB/g sediment. The risk assessor has decided that the area of influence is equal to about one tidal excursion based on the description of the local environment as moderately energetic. The state Department of Marine Fisheries provided local oceanographic information to calculate the tidal excursion lengths. The management area and its area of influence are collectively referred to as the disposal site area.

Modeling exposure point concentrations

Risk assessment is an iterative process, and initial calculations may not be sufficient to predict sediment or water-column concentrations. It may be necessary to use fate and transport models when the initial estimates of sediment or water concentrations at the management site or in the field of influence:

a. Exceed an obvious criterion, standard, or concentration which has a known toxicological significance.

b. Exceed some physical limit such as solubility or partitioning to a solid.

c. Result in a potential risk when carried through the risk assessment.

The USACE and USEPA provide significant support in those instances where sophisticated modeling is necessary to complete the exposure assessment. Models exist for predicting contaminant losses to air, surface water, and groundwater within the dredged material management program. The USEPA's Assessment and Remediation of Contaminated Sediments (ARCS) Program (USEPA 1996a) and the USACE Automated Dredging and Disposal Alternatives Management System (ADDAMS) (USACE 1995a) provide various models to estimate initial and longer term transport from a dredged material management site.

The ARCS program provides models which address contaminant losses:

a. During dredging, dredged material transport, and pretreatment.

b. Associated with specific management technologies such as confined disposal facilities, in situ capping and capped disposal, effluent and leachate.

c. From treatment trains such as thermal destruction, thermal desorption, biological treatment, extraction processes.

d. Due to the no action alternative.

ADDAMS is an interactive personal computer-based design and analysis system for dredged material management. The models include simple algebraic

expressions and numerical and analytical solutions to differential equations that are theoretically and empirically based. The USACE provides a technical note (USACE 1995a) which describes the available ADDAMS models, their application to various management technologies, a technical point of contact, and a request form for the models.

Output from the ADDAMS suite of models, which often provide contaminant flux rather than concentrations, can be used as input to a number of USEPA fate and transport models. These contaminant transport models are available from the USEPA Center for Exposure Assessment Modeling (CEAM). These contaminant transport models use mass balance principles and vary in complexity from simple analytical estimates which are useful to make initial calculations to numerically complex iterative models that predict time-varying contaminant fate and transport. These hydrodynamic and sediment transport models predict water and sediment concentrations. These include:

a. WASP4 - Predicts dissolved and sorbed chemical concentrations in sediment and overlying water. The model is time variable and can simulate three chemicals and three sediment size fractions simultaneously.

b. EXAMS II - This modeling system is also based on the WASP models. EXAMS predicts dissolved and sorbed chemical concentrations and can be run in a steady-state or quasi-dynamic mode. Unlike the WASP models, EXAMS does not simulate solids settling and resuspension.

c. SMPTOX3 - This is a simplified analytical steady-state model that calculates the distribution of contaminants in water and sediment. This model is typically used for initial calculations.

d. The product of this section is a description of the fate and transport model and its output. The description should include the equations which the model uses, the constraints on the model, the source of the model (e.g., USACE, USEPA), the input parameters, and any modifications which may have been made.

Step 3: Food chain modeling

The final step in the exposure assessment is to predict the amount of the contaminants of concern which a receptor will ingest, contact, or concentrate in its body. The risk assessment must express this exposure in the same manner as the available toxicological information. There are essentially three expressions of biological exposure:

a. Dose - amount of a contaminant of concern ingested per unit body weight of the receptor per day.

b. Body burden - concentration of a contaminant of concern per unit body weight or per unit body lipid.

c. Dietary concentration - concentration of a COC in the prey organism of a receptor.

The choice of which expression of exposure to use depends on the toxicity data available for a particular receptor.

For example, if the effects level for a given receptor is expressed as a dietary concentration (the concentration of a contaminant in the food of a receptor), then a dietary concentration associated with exposure at the disposal site should be calculated.

The calculation of doses, body burdens, and dietary concentrations proceeds in a similar manner to the prediction of exposure point concentrations. That is, the assessment may make an initial estimate based on relatively simple and reasonably conservative assumptions. The risk assessment must use a more sophisticated food chain model if the initial estimates:

a. Result in potential risk.

b. Ignore an essential exposure route defined in the conceptual model.

c. Exceed some known biological or physical limitation governing body burdens.

This is not to suggest continuous iterations. Rather, the risk assessor must ultimately choose a model which most realistically reflects site conditions and uses as much site specific information as possible.

Initial estimates of concentrations in infauna or fish

This text provides a simple calculation to estimate the concentrations of some COC in infauna and fish which may inhabit the management area and the local field of influence.

There are five classes of contaminants for which concentrations in infauna and fish may be important in the exposure assessment. These include: metals (generally only mercury biomagnifies), chlorinated organics (i.e., pesticides, PCBs, dioxin/furans), and polycyclic aromatic hydrocarbon (PAH). At dredged material management sites which have progressed to Tier III and Tier IV evaluations, the 28-day bioaccumulation results modified according to Clarke and McFarland (1991) to account for steady state provide estimates of invertebrate tissue concentrations. The risk assessment may use these tissue concentrations as input to food chain models to develop body burdens in higher trophic levels such as fish or piscivorous birds.

If a measured estimate of tissue concentration is not available for a COC, one can estimate concentrations of bioaccumulative compounds in biota (invertebrates or fish) based on a biota-sediment accumulation factor (BSAF) which expresses the accumulation of contaminants from sediments to the biota. The BSAF depends upon the concentration of the contaminant in the biota, Ca, the fraction

lipid of the biota (Fl), the concentration in sediments (Cs) and the fraction organic carbon of the sediments (Foc). The relationship is:

$$BSAF = (C_a/F_l)/(C_s/F_{oc}) \quad (1)$$

The final concentration of a bioaccumulative compound in wet-weight fish tissue, Ca, is expressed as the bioaccumulation through the sediment pathway as:

$$C_a = (C_s/F_{oc})\ H BSAF\ HF_l \quad (2)$$

where

C_s = average sediment exposure concentration for biota (calculated, see below)

BSAF = as calculated from site-specific data; data from the Tier III testing; or literature values (site-specific data are preferable)

F_l and F_{oc} are defined as above.

The term, C_s, can be calculated as:

$$C_s = C_d\ HF_a + C_o\ H(1-F_a) \quad (3)$$

where

C_d = 95th-percent upper confidence interval of the arithmetic average sediment concentration in the disposal site (projected or measured)

C_o = 95th-percent upper confidence interval of the arithmetic average sediment concentration outside the disposal site (measured)

F_a = fraction of time the organism spends foraging in the disposal site area.

Note that Fa will be 1 when the foraging area is equal to or less than the disposal site area and the area of influence. When the foraging area is greater than the disposal site area:

$$F_a = A_d/A_f \quad (4)$$

where

A_d = area of the disposal site and area of influence

A_f = foraging area of the receptor.

The ratio, F_a, may have to be adjusted based on site-specific data. For example, disturbance at the disposal site may increase the attractiveness of the site for a foraging species, causing A_f to approach the value of A_d.

This calculation assumes that the most likely bioaccumulative compound exposure pathways for fish are food-to-fish and sediment-to-fish pathways. This assumption holds only for those compounds in which:

a. Food ingestion, direct ingestion of sediment, and possibly gill contact with suspended sediment are the most important exposure mechanisms.

b. There is preferential binding to the sediment due to their hydrophobic properties.

c. Exposure to water-column foraging fish is extremely low due to the low solubility of these compounds.

The USACE provides bioaccumulation data (BSAF Database), which is downloadable from *http//www.wes.army.mil/el/dots/database.html*).

The product of this subsection is an initial estimate of the body burden of the COCs in a selected receptor.

Example 12: Estimating a Body Burden in Winter Flounder

The dredged material management area and its area of influence (defined previously as the area within one tidal excursion of the site) is approximately equal to one-half the summer foraging area of the winter flounder, based on observations made by the state's Department of Marine Resources. This species is a selected receptor, based on its commercial importance.

The proposed site is within the State Statistical Fishery Area 4, and is 2 percent of that area.

As indicated earlier, the upper 95th-percent confidence limit of the arithmetic average total PCB concentration in the sediments from the proposed dredging project area is
1ug total PCB /g sediment.

The upper 95th-percent confidence limit of the arithmetic average of total PCB in sediments at the reference site is 0.10 ug total PCB/g. The assessment assumes that this is the exposure point concentration for winter flounder when foraging away from the site and its area of influence.

The average fraction lipid of a flounder is 0.1, based on hypothetical data provided by a fisheries agency.

Therefore, the average sediment exposure concentration of total PCB, Cs, at the disposal site is:

$$C_s = (1 \times 0.5) + (0.1 \times 0.5) = 0.55 \text{ ug total PCB/g sediment}$$

The State has also supplied data indicating that the fraction organic carbon in sediments in the area is 0.05 (5 percent).

A locally calculated BSAF is 3, based on EPA studies of PCB in flounder and sediment in this bay. The projected body burden (weight wet), Ca, to a flounder exposed to this total PCB concentration in sediments of 5 percent organic carbon is:

$$C_a = (0.55/Foc) \times BSAF \times (Fl)$$
$$= (0.55/.05) \times 3 \times 0.1$$
$$= 3.3 \text{ ug total PCB/g wet weight flounder tissue}$$

This body burden value can be used in both human health and ecological risk assessments.

This example could have used a different species such as lobster. In that case, the general method would remain the same, but parameters such as foraging area, bioaccumulation factor, and fraction lipid would differ. Also, the example is relatively simple in that it does not address differential uptake and storage of PCB congeners among tissues. In some instances, it may be important to estimate uptake in organs other than muscle. For example, lobster hepatopancreas has a different fraction lipid than lobster muscle. In a human health risk assessment, where some individuals in a population may consume the hepatopancreas, it becomes important to calculate a separate concentration for that tissue based on its particular lipid content.

Use of Higher-Level Food Chain Models

In some cases, the risk assessment may require a more sophisticated food chain model which addresses exposure through food, water, and sediments. These models (summarized in Appendix B) often address a group of species and allow calculation of exposure concentrations through more complicated food chains. It is difficult to provide simple guidance regarding when the risk assessor should consult such a model. However, the complexity of the food chain models used in risk assessment will generally increase as:

a. The number of contaminants of concern increases.

b. The number of receptor species increases.

c. Higher trophic levels are a focus of concern.

d. The potential area affected by the dredged material management site increases.

e. The number of potential dredged material management options increase.

f. The number of exposure pathways increases.

The product of this step is an exposure dose, a dietary concentration, or a body burden calculated under the assumptions of a site-specific scenario. Subsequent sections will compare these to doses, dietary concentrations, or body burdens which are associated with a potential ecological or biological effect.

Ecological Effects Assessment

What is an ecological effects assessment?

An ecological effects assessment is a summary of the available data that describe the potential adverse biological effects of the COC on the selected receptors or closely related organisms.

What is the goal of the ecological effects assessment?

The goal of the ecological effects assessment is to provide the risk assessor with a description of the potential ecological effects associated with a COC and a concentration in environmental media, dose, body burden, or dietary concentration related to these effects.

What are the components of an ecological effects assessment?

The ecological effects assessment includes:

a. An identification of data sources.

b. A summary of ecotoxicological data.

c. A selection or calculation of a toxicity factor (i.e., concentration in environmental media, dose, body burden, or dietary concentration associated with a particular effect) which relates to the assessment end point chosen during problem formulation.

d. A description of the environmental effect associated with the toxicity factor.

How is the ecological effects assessment used in risk assessment?

Ultimately, the risk assessment will compare the toxicity factor developed in the ecological effects assessment to the predicted toxicity factor to predict risk.

Ecological Effects Assessment

The ecological effects assessment provides a description of the potential ecological effects associated with a contaminant of concern and selects a toxicity factor or factors (i.e., environmental concentration, dose, body burden, or dietary concentration associated with a particular effect). Figure 8 shows the general method for selecting and developing toxicity factors. Ultimately, the risk assessment will compare the toxicity factor developed in the ecological effects assessment to the predicted environmental concentration, dose, body burden, or dietary concentration from the exposure assessment to predict risk. The effects assessment proceeds in the following:

a. Identifying information sources.

b. Summarizing toxicological data.

c. Selecting and developing toxicity factors.

Step 1: Identify information sources

The first step in the effects assessment is to identify the data sources which may provide information on ecological effects and toxicity factors. The risk assessor should not rely on previously summarized information. It is important to update the ecological effects assessment for each COC within the risk assessment because the scientific literature is constantly adding to the database.

The effects assessment obtains such updated information from the technical literature and updates to USACE technical resources, USEPA and state guidance, and reports and publications of USEPA's Office of Research and Development. Appendix C summarizes a wide variety of information sources and WEB sites which provide information on toxicity of contaminants.

EPA's AQUIRE database should always be consulted as a primary source of toxicological information. On-line databases include: Bios Previews; Life Sciences Collection; Zoological Record Online; Enviroline; Pollution Abstracts; Oceanic Abstracts; and CAB Abstracts. Also, the TOXNET (TOXicological NETwork) and IRIS (Integrated Risk Information System) databases can be accessed via the National Library of Medicine's MEDLARS system. The U.S. Army Engineer Waterways Experiment Station's WEB page provides an Environmental Residue Effects Database.

The effects assessment should clearly identify the information sources consulted in its attempt to identify the known ecological effects associated with the COCs.

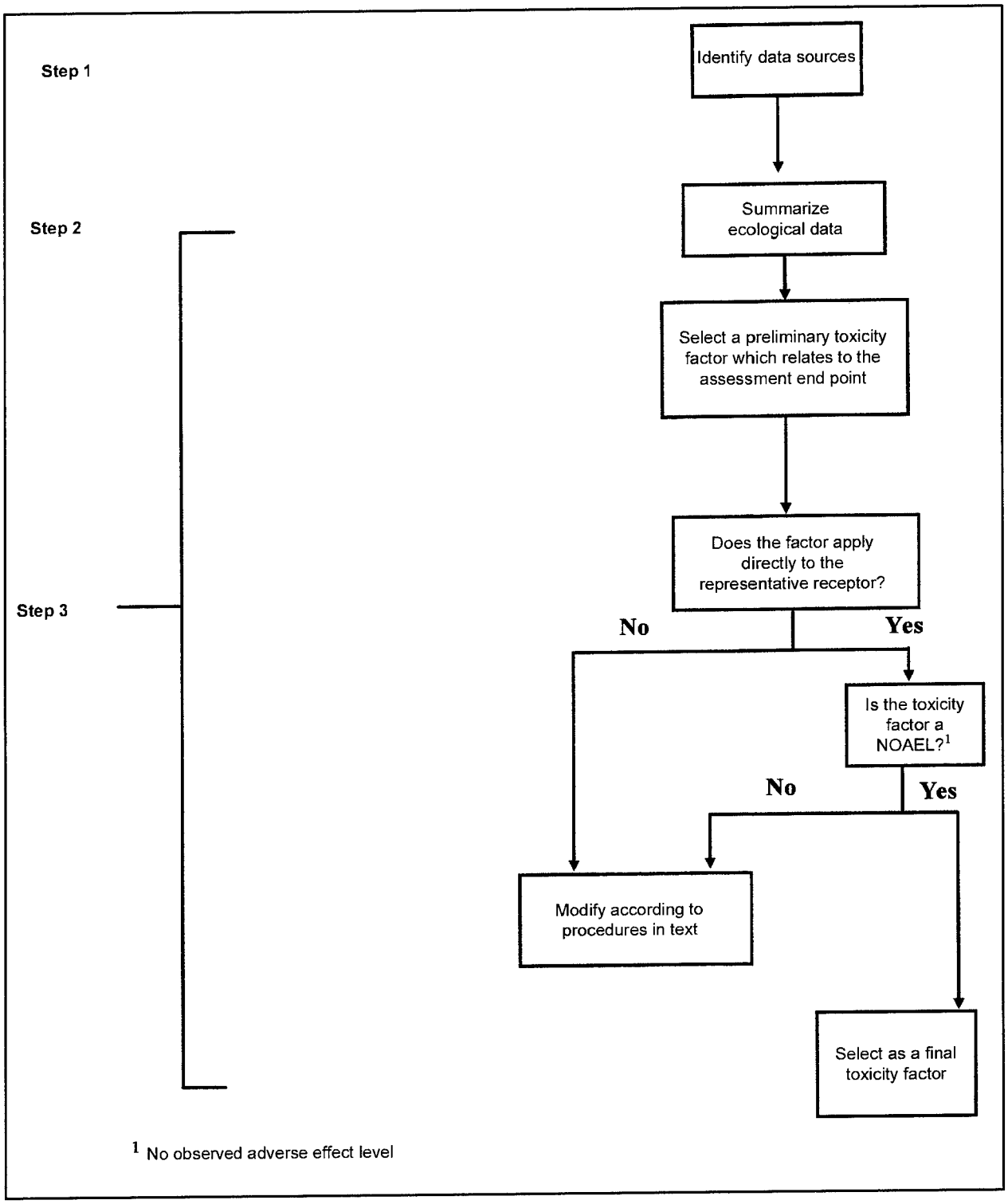

Figure 8. Steps in selecting and developing toxicity factors

Step 2: Summarize toxicological data

This section summarizes currently available toxicological data and provides toxicity factors as appropriate for the expressions of exposure. That is, the toxicity factors must be expressed in the same manner as the exposures. For example, exposures which are expressed as doses must have corresponding toxicity factors also expressed as a dose.

The summary should identify the toxic end points (i.e., the effect associated with each toxicity factor). The end points may include: lethality, reproductive impairment, behavioral modifications, or various sublethal toxic effects. End points may also include secondary effects such as loss of habitat. (As of this writing, the most commonly observed end points for aquatic receptors are lethality and reproductive impairment).

The types of toxicity factors often used include:

a. *Lethal effects*: Lowest reported or estimated nonlethal dose.

b. *Reproductive or developmental effects*: Lowest reported or estimated No Observed Adverse Effect Level (NOAEL - the concentration, dose, or body burden at which studies report no observed adverse effects) for reproductive or developmental effects. Effects can include: reduction in eggshell thickness, malformations of young, decrease in number of larvae or young produced, embryotoxicity, and reduction in number of eggs.

c. Systemic effects: Lowest reported or estimated NOAEL. Examples include: reduction in growth, hepatic enlargement, and other anatomical alterations considered adverse.

Appendix D provides detailed toxicological profiles for the likely contaminants of concern at dredged material management sites. The risk assessment should include a toxicological profile for each COC. These should be updated based on a query of information sources described in the text detailing Step 1.

Toxicological information may be derived from literature studies, Tier III and Tier IV bioassays, in situ bioassays, and field studies. Each method has inherent strengths and limitations. Information provided by various methods may include:

a. Concentrations or levels at which a COC elicits an adverse response in an individual organism or, where possible, a population.

b. A description of how the response of a test organism varies with the dose of a contaminant of concern (i.e., dose/response relationships).

c The type and magnitude of the response.

d. The identification of toxic end points.

Step 3: Selecting and developing toxicity factors

The selected toxicity factors must meet two general criteria:

a. They must relate to the assessment end point chosen during problem formulation.

b. They must be specific to the receptor species identified during problem formulation.

To meet the first of these criteria, the risk assessor must compare the toxic end point and described effect to the assessment end point. For example, if the assessment end point was protection of a commercial fishery, the toxicity factor must have an end point and described effect which relates to the maintenance or reproductive success of the species of commercial interest. A toxicity factor associated with reduced reproduction in fish applies, but a factor which may indicate eggshell thinning in shore birds is not applicable in this example. The two toxicity factors may be very different in magnitude, but only the value appropriate to the assessment end point applies. This is why it is so important to have an updated summary of the toxicity factors and their associated effects. It is only through this description that the risk assessor can judge whether a particular toxicity factor is applicable to the assessment end point.

This subsection provides several methods to calculate toxicity factors. The effects assessment should attempt to identify or develop toxicity factors for the selected receptors. If a receptor-specific toxicity factor is available, the risk assessment should use it. However, in many instances, such receptor-specific information will not be available from the literature or the sediment evaluation procedures, and the risk assessor will have to develop a toxicity factor based on information from other species. In such instances, the risk assessor may extrapolate from related information.

This subsection provides several methods for making these extrapolations.

The product of this section is a summary of available toxicological data and a selection of a toxicity factor for each COC. The selection should include the reason for selecting the particular toxicity factor and an explanation of how it relates to the receptor of concern and the assessment end point.

Example 13: Selection of a Toxicity Factor for Exposure of Winter Flounder to Total PCBs

Black et al. (1998) assessed the effects of PCBs on the reproduction of a fish using Fundulus heteroclitus (marine minnow) as an experimental organism. They measured a Lowest Observed Adverse Effect Level (LOAEL) at 3.8 ug PCB/g wet weight and an NOAEL of 0.76 ug PCB/g wet weight. The risk assessor chose a body burden of 0.76 ug PCBs/g wet weight as the toxicity factor. This is an appropriate toxicity factor because:

a. It addresses toxicity to total PCBs, the COC.

b. It is from a study which includes the measurement of an NOAEL as well as an LOAEL.

Black et al. describe the end points in the study as female mortality and decreased egg production, therefore, the toxicity factor relates to the assessment end point "Health and Maintenance of the Local Flounder Population."

Gas Research Institute (GRI) approach for developing toxicity factors

The Gas Research Institute (GRI 1996) has developed a protocol for selecting or developing toxicity factors for a COC. It includes the following:

a. Select a value if an appropriate state or Federal agency has proposed it.

b. In the absence of a proposed value and if data are available on NOAELs for the receptor species or for species that are phylogenetically and ecologically similar to the selected receptor species (e.g., from the same family of birds or mammals), select the lowest NOAEL.

c. If NOAELs for phylogenetically similar species are unavailable, the assessment adjusts NOAEL values for other species (as closely related as possible) by dividing by a factor of 10 to account for extrapolations between families or orders. The lowest NOAEL is used whenever several studies are available. This interfamily extrapolation is similar to EPA's derivation of human health reference dose (RfD) values, where animal studies are extrapolated to humans by dividing by a factor of 10.

d. In the absence of appropriate NOAELs, if LOAELs (the lowest concentra-tion, dose, or body burden available in the literature at which an effect occurs) are available for phylogenetically similar species, divide these by a factor of 10 to account for an LOAEL-to-NOAEL conversion. The LOAEL to NOAEL conversion is similar to EPA derivation of human health RfD values, where LOAEL studies are adjusted by a factor of 10 to estimate NOAEL values.

e. For calculating chronic toxicity values from data for subchronic tests (e.g., acute data), the resultant LOAEL or NOAEL values are divided by

an additional factor of 10. This is consistent with what is done in deriving human health RfD values.

f. In cases where NOAELs are available as a dietary concentration (e.g., milligram contaminant per kilogram food), a consumption rate for marine birds or marine mammals may be estimated based on various food intake summaries (e.g., USEPA's Wildlife Exposure Factors Handbook (USEPA/Office of Research and Development 1993)) and a corresponding NOAEL may be calculated. This consumption rate is expressed as a percentage of the animal's body weight on a wet weight basis or in units of kilogram of food (wet weight) per kilogram of body weight per day (kg/kg/day).

g. Some NOAEL values may be over conservative because they provide information on which dose produces no effect, but not how much higher the concentration has to be to produce an effect. Where the lowest NOAEL available in the literature is so low that background concentrations will produce a dose that exceeds it, reject the lowest NOAEL and use the next highest NOAEL.

California EPA approach

The following description is adapted from the California EPA approach for calculating toxicity factors (adapted from California EPA 1996).

a. Use toxicity data for representative species and members of the same taxonomic family in estimating toxicity to representative species.

b. If data are lacking or judged inappropriate, use data for surrogate species following application of one or more uncertainty factors (UFs). These UFs may be based on data when available or, in the absence of data, on the default values provided below.

(1) Apply a UF of 500 to adjust from less sensitive end points, such as mortality, to a chronic NOAEL (e.g., LD50 to NOAEL Chronic).

(2) Apply a UF of 10 to adjust from an acute LOAEL to a chronic NOAEL (e.g., LOAEL Acute to NOAEL Chronic).

(3) Apply a UF of 5 to adjust from LOAEL to NOAEL.

(4) Apply a UF of 1 for interspecies extrapolations within the same taxonomic family (e.g., beagle to fox - canidae to canidae).

(5) Apply a UF of 5 for interspecies extrapolation within the same taxonomic order.

(6) Apply a UF of 10 for interspecies extrapolation between taxonomic orders.

California DEP notes that these UFs are in the range of chronic and subchronic NOAEL comparisons in studies of uncertainty factors currently in preparation by USEPA and other discussions of uncertainty factors.

EPA Region X approach

EPA Region X provides an approach for calculating toxicity factors (from EPA Region 10, 1996 and based on Sigal and Suter 1989).

The features of this approach follow.

a. Apply a UF of 10 to convert from an acute or subchronic LOAEL value to a NOAEL value.

b. Apply a UF of 5 to convert from a chronic LOAEL to a chronic NOAEL value.

c. Apply a UF of 2 for interspecies extrapolations among families within the same order for nonprotected species.

d. Apply a UF of 2 for interspecies extrapolations among orders within the same class for nonprotected species.

e. Apply a UF of 2 to convert a NOAEL for a nonprotected species to a related protected species.

The investigator should determine which approach is most appropriate for a site. Often, this is based on geography inasmuch as different states or regions may have developed different approaches for accounting for uncertainty.

The use of toxicity models

There are currently several efforts to develop models which may aid in the assessing the toxicity factors in a comprehensive and additive manner. Examples include the summed PAH model (Swartz et al. 1995) which attempts to predict the toxicity of mixtures of PAH compounds using the concept of toxic units (Appendix B). This model attempts to predict the probability of significant acute toxicity to benthic infauna from exposure to sediment concentrations of a mixture of PAHs. The obvious current limitation is that it does not address chronic effects: the critical body residue or narcosis models (e.g. McCarty et al. 1992; McCarty and Mackay 1993) which attempt to assess the acute (and in some cases chronic) toxicity of mixtures of hydrophobic neutral narcotic chemicals. This model is appropriate for use when the exposure is expressed as a body burden.

Risk Characterization

This section describes the general methods used to make qualitative and quantitative characterizations of risk. These include the use of the toxicity

quotient method and the application of a weight of evidence approach recently developed in the state of Massachusetts.

Risk characterization is an integration of the exposure assessment and effects assessment to judge whether the predicted exposure to the COC are of sufficient magnitude to produce the effects associated with the selected toxicity factor.

The assessment should characterize risks with respect to the stated assessment end points. This requires integrating exposure and effects information specific to that assessment end point.

For each assessment end point, the risk characterization should:

a. Estimate the area(s) within which receptors or habitats are considered to be at risk.

b. Provide an estimate of the magnitude of the risks within these areas.

c. Provide information on the persistence or duration of these estimated risks.

d. Identify the pathways and other conditions which contribute to the risk.

e. Identify and characterize the uncertainties associated with the risk estimates.

The risk characterization integrates effects and exposure information in one or more of several methods, including quotient methods, weight-of-evidence or lines-of-evidence approaches, and probabilistic methods.

Generally, risk characterization uses a direct numerical comparison between the exposure concentration, dose, body burden, or dietary concentration and their associated toxicity factors. If the ration between them is greater than one, there is potential for risk. In those instances where an assessment end point has several measurement end points (and hence several toxicity factors to compare with each measurement end point), risk characterization may use a weight-of-evidence approach.

Quotient Method

The Quotient Method is a simple tool for comparing exposure concentrations to toxicologically effective concentrations:

$$HQ = EPC/TF \qquad (5)$$

where

HQ = hazard quotient

EPC = exposure point concentration, dose, body burden, or dietary concentration reflecting exposure for relevant exposure areas; these may be point estimates or summary statistics; this is expressed in the same units as the TF

TF = the selected toxicity factor appropriate for the chemical and receptor.

HQs in excess of "1" are indicative of potential risk. Because these are often based on threshold TF values, it is difficult to judge the magnitude of risk. Nevertheless, the degree to which TF exceeds "1" provides a qualitative indication of magnitude. Quotient methods can be utilized in weight-of-evidence and probabilistic approaches. For the latter, distributions of TF and EPC values can be derived (Suter et al. 1993).

Weight-of-Evidence or Lines-of-Evidence Approaches

The risk assessment can apply weight-of-evidence approaches when relating multiple measurement end points to an assessment end point. Typically, these approaches consider:

a. The weight or level of confidence given to the individual measurement end points used to evaluate the assessment end point based on strength of association between assessment and measurement end points, data quality, and study design and execution as described earlier in connection with selecting the measurement end points.

b. The magnitude of response of each measurement end point based on absolute magnitude, spatial extent, and duration.

c. Concurrence among the measurement end points (i.e., if all the measurement end points agree, this increases the weight of the overall assessment).

These three elements permit the investigator to assess the overall weight of evidence or to resolve information that may be disparate. The USEPA espoused weight of evidence but provides no guidance for executing an approach. Menzie et al. (1996) provide a quantitative and qualitative method based on the efforts of a workgroup comprised of industry and government representatives. Sample, Opresko, and Suter (1996) developed a qualitative approach. Both the weight-of-evidence or lines-of-evidence approaches underscore the importance of being open, consistent, and less subject to hidden biases.

Example 14: Risk to Flounder

The appropriate method to assess risk to flounder is to compare a measured effect level for body burden of PCBs in flounder to the calculated flounder body burden. As indicated earlier, the selected toxicity factor is 0.76 ug PCB/g wet weight. This is less than the 3.3 ug PCB /g body tissue concentration calculated for winter flounder in this example. Therefore, the assessment shows that there is potential for risk to the selected receptor, winter flounder. At this point, the risk assessor and risk mangers can:

a. Accept the initial conclusion and employ risk management activities.

b. Employ more complex fate and transport models and perhaps a more complex food chain model and recalculate risk.

The conclusion of risk from the initial estimates has various sources of uncertainty including:

a. Uncertainty concerning the actual foraging area of a flounder

b. Uncertainty concerning the BSAF — the assessment used the recommended BSAF of 3 which may be overly conservative. A more sophisticated food chain model may give a more realistic estimate of body burden.

c. Uncertainty associated with possible interspecies differences between the experimental organism, Fundulus heteroclitus, and the flounder.

d. All the models used in the assessment are linear. Therefore, a simple sensitivity analysis can be performed using the ranges of various parameters.

Note that this estimate of potential risk applies to PCB exposures. The risk from the other COCs at this hypothetical site (PAHs and mercury) should be estimated as well. Also the risk characterization is iterative. At this point, the risk assessor may want to implement more sophisticated estimates of sediment concentrations using data intensive modeling. The assessor may also use a more sophisticated food chain model (e.g., Appendix B).

What Is a Human Health Risk Assessment?

A human health risk assessment is an estimate of potential health risk to individual humans who are exposed to contaminants of concern while conducting specific activities.

What Are the Components of a Human Health Risk Assessment?

The human health risk assessment integrates four general components in making a risk estimate. These include:

a. Hazard identification - an initial description of potential health effects associated with the contaminants of concern and an estimate of acute risk if such is likely.

b. Exposure assessment - an estimate of the dose of a contaminant received by an individual human under specific conditions and while conducting specific activities (detailed within the exposure assessment).

c. Toxicity assessment - a summary of the human health effects associated with each contaminant of concern and a choice of an appropriate end point (toxicity factor) against which to judge potential risk.

d. Risk characterization - an estimate of potential risk to individuals based on a comparison of the dose calculated in the exposure assessment to the end points defined in the toxicity assessment.

What Are the Criteria for Judging Human Health Risks?

Human health risks depend on an estimate of the potential for carcinogenic risk and noncarcinogenic risk for each contaminant. The potential for carcinogenic risk depends on an estimate of the carcinogenic potential of a contaminant (expressed as a probability of increased cancer risk) and the noncarcinogenic risk based on a comparison of a threshold dose for a contaminant of concern to the dose calculated in the exposure assessment.

4 Human Health Risk Assessment

This section provides guidance for developing human health risk information for exposures to contaminated sediments related to the disposal of dredged material.

This guidance follows USEPA human health risk assessment guidance documents and manuals. Individuals conducting or evaluating human health risks should be familiar with the guidance contained in:

a. Risk Assessment Guidance for Superfund, Volume I, Human Health Evaluation Manual (Part A), Interim Final (USEPA/Office of Emergency and Remedial Response (OERR) 1989a)

b. Assessing Human Health Risks from Chemically Contaminated Fish and Shellfish (USEPA/Office of Marine and Estuarine Protection and Water Regulations and Standards (OMEP) 1989)

c. Addendum to Methodology for Assessing Health Risks Associated with Indirect Exposure to Combustor Emissions (USEPA/Office of Health and Environmental Assessment (OHEA) 1993-Review Draft)

d. Guidance for Assessing Chemical Contaminant Data for use in Fish Advisories. Volume I: Fish Sampling and Analysis (USEPA/Office of Science and Technology (OST) 1993)

e. Guidance for Assessing Chemical Contaminant Data for use in Fish Advisories. Volume II: Risk Assessment and Fish Consumption Limits (USEPA/OST 1994)

f. Methodology for Estimating Population Exposures from the Consumption of Chemically Contaminated Fish (USEPA/Offices of Policy, Planning and Evaluation, and Research and Development 1991)

The following subsections are organized to conform to the four basic components of human- health risk assessment: Hazard Identification, Exposure Assessment, Toxicity Assessment, and Risk Characterization.

Hazard Identification

The hazard identification section addresses the nature and extent of the problem. It should:

a. Identify contaminants of concern (Note that the problem formulation has already provided a list of contaminants of concern for human and ecological receptors).

b. Briefly summarize what is known about the capacity of contaminants of concern to cause cancer or other adverse effects in laboratory animals and in humans.

c. Describe whether there is the potential for bioaccumulation of these contaminants through the food web to a human receptor.

d. Where possible, identify contaminants in sediments which may act together (synergistically, antagonistically, or additively) as complex mixtures in exerting toxic effects in humans.

A human health risk assessment hazard identification should also assess the potential for exposure to concentrations in sediments which may result in acute toxicity. However, because dredged material disposal sites are generally offshore, acute exposure conditions are very unlikely.

Human Health Exposure Assessment

A human health exposure assessment builds upon the qualitative descriptions in the conceptual model to calculate a quantitative estimate of the exposure of selected human receptors to the COC. This quantitative estimate may be:

a. A concentration in some environmental media such as sediment or water.

b. An estimate of the dose of a contaminant of concern to a human receptor through ingestion of fish or shellfish.

What are the steps in conducting a human health exposure assessment?

The human health exposure assessment proceeds by:

a. Describing the exposure pathways along which humans may contact the contaminants of concern.

b. Consulting EPA guidance and background documents which provide information on various factors which may affect the calculation of human exposures to contaminants of concern.

c. Estimating the amount of a contaminant of concern which a human receptor may ingest or contact.

How does the exposure assessment relate to human health risk?

The human exposure assessment should quantify the exposure a dose of contaminant of concern for comparison to published human toxicity factors for cancer and noncancer effects.

Exposure Assessment

The exposure assessment develops exposure scenarios which are detailed descriptions of:

a. A human receptor's activities which result in exposure to the COC.

b. The pathway and route by which the human receptor contacts COC.

c. Physical, chemical, and biological factors which affect the amount of the contaminant contacted or ingested.

For each exposure scenario, the human health exposure assessment estimates human exposure to COCs in the dredged material at the dredged material management site. The risk assessment may develop present and likely future exposure scenarios, depending upon site-specific characteristics. For example, a newly proposed disposal site may require only an assessment of future risk, while an existing disposal site for which a new source of dredged material disposal is planned may also require an analysis of present exposure and risk as well.

The exposure assessment requires iterative steps to characterize the potentially exposed receptors (Figure 9). These steps are integrated into the site-specific conceptual model begun during problem formulation, and include:

a. Consulting current EPA guidance and background documents.

b. Quantifying the exposure.

c. Describing the receptors and exposure pathways.

The products of the Exposure Assessment are a conceptual model of the site, which demonstrates the links between contaminated media and humans, and a quantitative estimate of the exposure concentration and doses for the individual defined in the exposure scenarios. There are typically several exposure scenarios considered for each assessment.

Step 1: Consult USEPA resource documents

There are several USEPA publications that assist in developing the exposure scenarios. These documents provide such information as how often people eat seafood, how much seafood is ingested per meal, how much of a particular contaminant may be absorbed upon ingestion or dermal contact, etc. The risk assessment uses these factors in calculating exposure to the contaminants of concern. The following USEPA documents should be consulted as an integral part of the human health exposure assessment.

a. "Exposure Factors Handbook" (USEPA 1989).

b. "Exposure Factors Handbook" (USEPA/ORD 1995).

c. "Human-Health Evaluation Manual, Supplemental Guidance: Standard Default Exposure Factors" (USEPA/OSWER 1991a).

d. "Consumption Surveys for Fish and Shellfish. A Review and Analysis of

e. "Final Guidelines for Exposure Assessment" (USEPA 1992c).

f. "Guidance for Assessing Chemical Contaminant Data for Use in Fish Advisories. Volume I: Fish Sampling and Analysis" (USEPA/OST 1993).

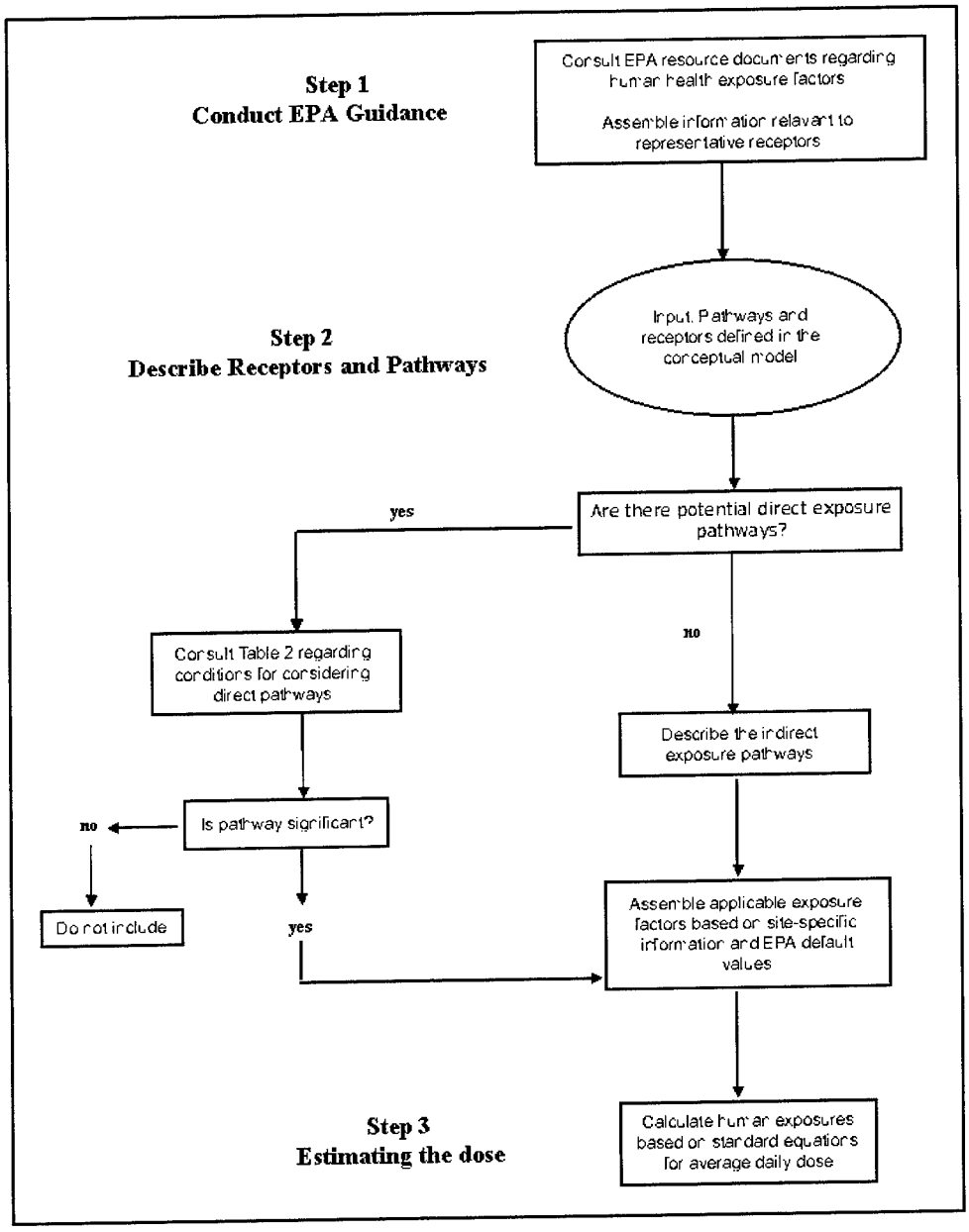

Figure 9. Developing a human health exposure assessment

g. "Guidance for Assessing Chemical Contaminant Data for Use in Fish Advisories. Volume II: Risk Assessment and Fish Consumption Limits" (USEPA/OST 1994).

Step 2: Describing the receptors and exposure pathways

There are several potential pathways by which people may be exposed to contaminants in dredged material at a management site. Individual exposures occur either through direct or indirect exposure pathways. Potential direct exposure pathways include dermal contact and ingestion of contaminated sediments or surface water. Indirect exposure pathways include ingestion of

seafood (finfish and shellfish, from either marine or freshwater sources) which contains contaminants of concern. A complete exposure pathway must include:

a. A source and mechanism of release of contaminants.

b. A retention or transport mechanism for exchange of contaminants between media.

c. An exposure point (e.g., sediment, water) where contact occurs.

d. An exposure route (e.g. ingestion, dermal uptake) by which contact occurs (USEPA/OERR 1989).

Direct exposure pathways

In most dredged material management activities, the direct human exposure pathway is unlikely to be of concern. Therefore, the body of this guidance does not provide detailed information. In many cases, particularly for offshore disposal, direct human exposure to contaminated sediments at aquatic dredged material management sites is unlikely because the exposure pathways are incomplete. The direct pathways may be more likely at containment islands and nearshore management facilities. They may also occur during transport and handling of material.

Direct exposure to sediments. Although the sediments are a source of contaminants, there is no strong exchange mechanism between the sediments and the overlying water since the contaminants are sediment sorbed in most cases. This makes transport to the surface through desorption and dissolution unlikely for most contaminants. Direct exposure through the water column may be event mediated as in the case of storms or erosive events. Exposures due to direct contact with sediments through activities such as swimming, recreational activities, or fishing are also unlikely in offshore aquatic sites because:

a. Distance offshore and water depths at dredged material management sites are generally incompatible with recreational swimming.

b. Depth to the bottom makes direct contact with sediments by fishermen and boaters unlikely.

Direct exposure to water. There is potential for human contact with a waterborne plume near or at the dredged material disposal site immediately following disposal operations. However, the duration of this contact would be short, and the frequency of contact would be low because it would occur only during disposal operations. Therefore, this direct exposure pathway is likely to be insignificant. Disposal in nearshore environments may warrant consideration of direct exposure pathways.

When to consider the direct exposure pathways. There may be instances where direct exposure pathways are likely during a dredged material management activity. Whether to incorporate these pathways into the human health exposure

assessment depends on various site specific factors. Table 2 provides guidance on when these direct pathways may be of concern. Appendix E provides the equations to quantify these potential exposures.

Table 2
Conditions Which May Require Assessment of Direct Human Health Exposures

Direct Pathway	Potential Receptors	Conditions which May Require Assessment of Pathway
Direct ingestion of sediment	Recreational users (swimmers, waders, boaters, naturalist, trespassers) or off-site resident	• Nearshore site • Intertidal site • Containment island which may attract recreational users • Upland site (for a naturalist or trespasser).
Direct ingestion of sediment	Dredged material management workers (barge worker, pipeline worker)	• Dredged material management sites which require workers to be at the site for more than one season • Dredged material management options which may require routine contact with sediments • Dredged material management which may require long-term maintenance of a management facility.
Direct ingestion of surface water	Recreational or off-site resident	• Near shore site • Intertidal site • Containment Island • Upland site where groundwater discharge is a potential concern.
Direct ingestion of surface water	Worker	• Dredged material options which may require long-term maintenance of a facility • Upland sites where discharging groundwater or dewatering in excavation may occur.
Inhalation of volatilized contaminants or fugitive dust	Worker or off-site resident	• Management options which require dredged material to be exposed to atmosphere, especially nearshore • Management or transport options which allow dredged material to dry at surface during transport or storage.

Indirect exposure pathways

For aquatic disposal, the most likely human pathway is an indirect exposure pathway through consumption of fish or shellfish (Figure 10). Therefore, this section provides details and examples for assessing the pathway. Within this pathway, the likely exposure route for humans to contaminated sediments and surface water is the ingestion of fish or shellfish that have accumulated these compounds. This exposure pathway fulfills the criteria for a complete exposure pathway (as described above) because:

a. There is a source of contaminants — the sediments at the dredged material management site.

b. There is a transfer mechanism between the sediments and the seafood — bioaccumulation.

c. There are exposure points where contact occurs — the commercially or recreationally caught seafood which have been exposed to contaminants from the management site.

d. There is an exposure route — the consumption of this seafood.

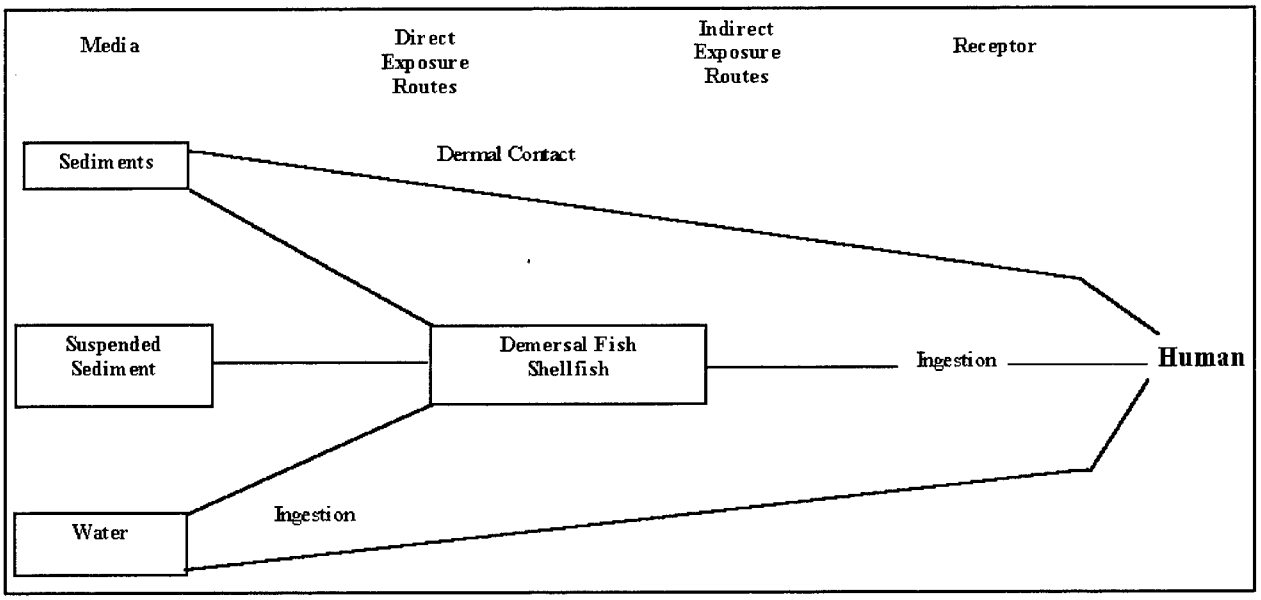

Figure 10. Example of a conceptual model showing direct and indirect exposure pathways for human health

Characterization of this exposure setting for seafood ingestion requires:

a. Defining the exposed human population.

b. Characterizing the individual's and population's activities and exposure route (i.e., consumption of seafood).

c. Identifying the species consumed.

Defining the exposed population. The assumed exposed population should be of individuals who potentially consume seafood that is exposed to the contaminants at the dredged material site. This may be a local population consuming seafood from a fishery which does not export outside a constrained geographic area. Alternatively, the fishery may be serving a large metropolitan area. When possible, efforts should be made to identify any sensitive populations, such as pregnant women and young children, and any groups that may be subject to disproportionately high exposures, such as subsistence fishermen [e.g., immigrant groups and Native Americans (Executive Order 12898)].

Characterize receptor activities. Different exposure scenarios used in a human health risk assessment may result in different risk estimates and different management responses to those risks. Therefore, it is important to fully and accurately characterize the types of activities which lead to exposure within each scenario. The activities and indirect exposure route that are addressed in this guidance include consumption/ingestion of seafood from:

a. Recreational or subsistence fishing.

b. Commercial fishing.

More than one exposure scenario for the ingestion of seafood may be required for full characterization of human receptors. There may be several fisheries potentially influenced by the disposal site, or the site may be used simultaneously by commercial and recreational fishermen. Sources of site-specific information that can be used to define the receptor's activities include:

a. Local and state departments of fisheries.

b. National Marine Fisheries Service (NMFS) National Oceanic and Atmospheric Administration (NOAA).

c. Local university fishery and/or wildlife departments.

d. Surveys of local residents and fishing groups.

e. Local seafood distributors.

Identify the species. The Exposure Assessment should identify the dominant species of seafood landed locally for recreational, subsistence, and commercially caught seafood because the concentration of the contaminants in the seafood will depend upon the foraging habits of the organisms, their ability to bioconcentrate the chemicals of concern, and their position in the food web.

Information on the species that are harvested and their biology is often available through surveys and catch statistics from the NMFS, local or state

departments of fisheries, or local universities. Local surveys of recreational fish catches and consumption information from these sources may also be available. Also, the dietary and cultural habits of the exposed populations can often allow the risk assessor to define the list of species likely consumed by recreational fishermen.

Indirect pathway - Recreation/subsistence catch. Many human health risk assessments assume that recreational or subsistence fishermen obtain all of their seafood from the disposal site. This is a very conservative assumption which assessors often make when using the subsistence scenario as a worst-case screening tool. At dredged material management sites, this guidance recommends modifying this conservative assumption to incorporate the seasonality of the catch and the receptor's preferences for different species of seafood. Additionally, the size of the disposal site relative to the recreational/subsistence fishing area should be evaluated.

Indirect pathway - Commercially harvested catch. For consumers of commercially harvested seafood, the risk assessment should assume that:

a. The human receptor's entire seafood diet is derived from seafood landed locally (i.e., within the state immediately inshore of the disposal site), unless there are available data to the contrary.

b. The amount of contaminated seafood in this diet is proportional to the amount of the catch influenced by the disposal site. For example, if one assumes that the receptor's seafood diet derives from a 20-square-mile bay inshore of the disposal site, and the site only influences 1 square mile, then the contaminated portion of the receptor's seafood diet is adjusted by 1/20 (see the example).

The first assumption is conservative (i.e., protective of human health) because it does not allow the seafood diet to be diluted by catch from distant areas. For the second assumption, the risk assessor will need to estimate the total landings relative to the landings influenced by the disposal site. This calculation will require data from state or Federal statistical reports which tabulate landings by fishing areas offshore of each coastal state.

In the absence of information for commercial catches, the recreational fishing exposure scenario should be used.

The product of this section is a narrative or tabular presentation of consumers of potentially contaminated seafood, that includes where the seafood is landed, what species of seafood are consumed, and any other information that describes an individual's or population's behavior relative to seafood consumption. This information will allow the risk assessor to calculate estimates of contaminant intake to the identified receptors.

Example 15: Description of Indirect Pathway - Consuming Winter Flounder

The management site is within a larger area representing a winter flounder commercial fishery. The site is close enough to shore to be a recreational fishery as well (although this example carries through only the commercial fishing scenario).

The flounder are landed at a medium-sized city on the local bay, and the consumers are the people in the local metropolitan area. The State Department of Marine Fisheries indicates that little, if any, of the flounder are exported to a larger area.

Step 3: Quantify exposure

The quantification of indirect exposure proceeds by:

a. Specifying the equation to calculate a dose.

b. Estimating the exposure point concentration.

c. Reviewing site specific information for exposure factors.

d. Reviewing EPA default assumptions.

e. Running the calculation.

The exposure assessment quantifies exposure to human populations using a set of fairly standard equations the choice of which depends upon the receptor, exposure pathway, exposure route, and receptor activities. The equations calculate a dose based on:

a. Exposure point concentrations.

b. Ability of the receptor to absorb the contaminant.

c. Ingestion rate.

d. Amount of seafood ingested from the area under the influence the management area.

e. Frequency of seafood meals.

f. Body weight of the receptor.

g. Time over which the receptor consumes seafood.

This section describes those equations and their use for the indirect pathway. Appendix E provides a set of equations to use for the less likely direct pathway.

Specifying the equation to calculate dose

After selecting and describing the exposure pathways, the exposure assessment must calculate the intake of the contaminant of concern (in milligrams contaminant per kilogram body weight per day). This is the dose of contaminants that enters the human body through the gastrointestinal tract following consumption of contaminated seafood. USEPA guidance describes this dose as the Average Daily Potential Dose (ADD_{pot}). This is a central calculation in the human health assessment because it integrates all the elements of the exposure assessment. For the assessment of risk associated with contaminated dredged materials, it should be calculated for each of the individual fish species that are ingested by each receptor described in the exposure scenarios.

Figure 11 shows the elements in this equation and their sources. The quantification of this exposure is expressed as the product of the exposure point concentration and various exposure factors:

$$ADD_{pot}\left(mg/kg/day\right) = \frac{EPC \times Abs \times IR \times FI \times EF \times ED}{BW_{avg} \times AT} \qquad (6)$$

where

EPC	=	Exposure point concentration in seafood influenced by the dredged material disposal site (mg/kg)
Abs	=	Fraction of contaminant absorbed from the seafood through the gastrointestinal tract
IR	=	Ingestion rate (kg/meal)
FI	=	Fraction of seafood ingested from contaminated source (unitless)
EF	=	Frequency of potential exposure events, total annual seafood meals ingested (meals/year)
ED	=	Duration of the exposure period (years)
BW_{avg}	=	Average body weight of receptor (kg)
AT	=	Averaging time (days)

According to USEPA guidance (USEPA/OERR 1992a,b), the EPC and the exposure factors in this equation should represent reasonable maximum exposures (RME). The RME is a plausible estimate of the individual risk for those individuals at the upper end of the risk distribution. Under the reasonable maximum exposure case, a combination of 50^{th}- and 90^{th}-percentile values of exposure factors should be used for intake rates, fraction of seafood diet harvested from the disposal site, exposure frequency, and exposure duration. Table 3 summarizes the factors risk assessors need to consider when determining default values or directly measuring values for this calculation.

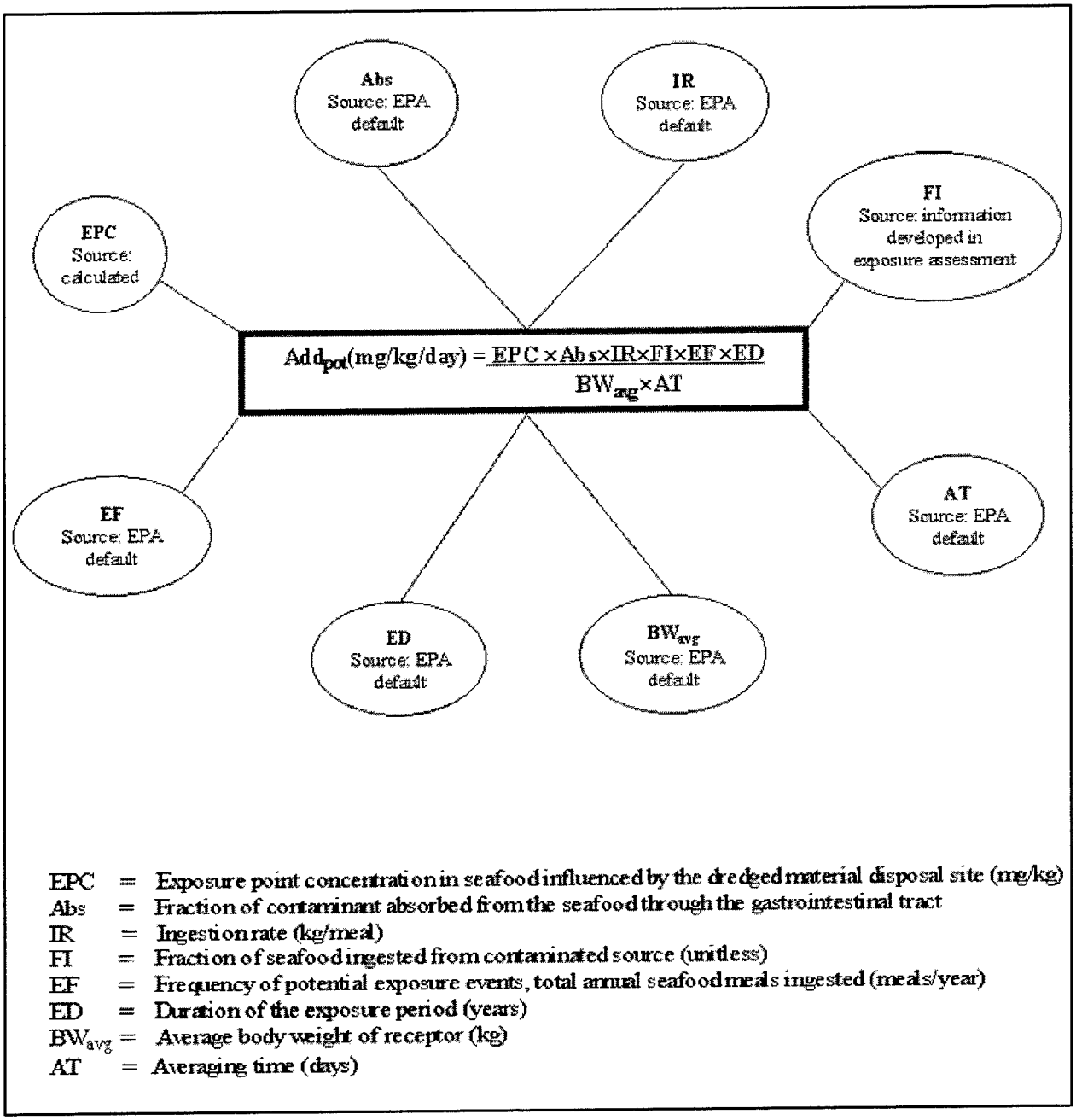

Figure 11. Factors for calculating average daily dose and the source for each factor

Estimate exposure point concentrations (EPCs)

EPCs are the contaminant concentrations in the edible tissue of seafood from the dredged material disposal site. The most reliable method for determining the EPCs in the species of concern is by directly measuring the concentrations in the tissues of the organisms. However, this is typically not an option, unless the seafood species of interest can be collected from the dredging (project) site and their foraging area is confined to that area or its area of influence.

Table 3
Uncertainties Associated with Calculating the Average Daily Potential Dose

Equation from text: $$ADD_{pot}(mg/kg/day) = \frac{EPC \times Abs \times IR \times FI \times EF \times ED}{BW_{avg} \times AT}$$

Term	Description	Default	Considerations/Uncertainty
ADDpot	Average Daily Potential Dose	Calculated value	
EPC	Exposure point concentration	Site-specific data; calculated or measured	Measure seafood tissue contaminant concentrations if possible, or calculate as detailed in text on Hazard Identification.
Abs	Fraction of contaminant absorbed from the seafood through the GI tract	"1"	Depends on lipid composition and preparation of seafood consumed.
IR	Ingestion rate	Site-specific data	Depends on the behavior of the seafood consuming population.
FI	Fraction ingested	"1" (subsistence and recreational fisherman)	See text for estimations of FI for commercial catch.
EF	Exposure frequency	Approximate range of 10 to 100 meals/year	Varies, depending on the behavior of the seafood-consuming populations.
ED	Exposure duration	9 years (median) 30 years (upper-bound)	Use site-specific data, especially if time of capping of dredged material disposal is known.
BW	Body weight	70 kg (adults) 15 kg (children)	Intermediate values should be used for teenagers. Values for infants will be lower than 15 kg.
AT	Averaging time	70 years	

In the absence of measured data for EPC, the risk assessment uses the tissue concentrations of contaminants in the seafood estimated in the ecological risk assessment. The EPC obtained from the application of these methods should be expressed in milligrams (mg) of contaminants per kilogram (kg) of seafood.

The product of this step is an estimate of the concentration of COCs in seafood exposed to the management area and its area of influence.

Example 16: Body Burdens in Winter Flounder

As indicated earlier, the risk assessor has identified a population in the area potentially exposed to PCBs from flounders in a commercial catch. The proposed disposal site will influence a fraction of this flounder catch. As described earlier, a tissue concentration of total PCBs can be calculated for flounder, based on measured sediment concentrations and observed biota-to-sediment concentration factors. These calculations resulted in a wet weight tissue concentration of 3.3 ug total PCB/g flounder tissue for flounders foraging over the disposal site. This is the EPC for total PCBs in the human health risk assessment.

Reviewing site-specific information for exposure factors

Wherever possible, exposure factors should be developed from site-specific information. For example, local knowledge of subsistence fishermen may provide a site-specific ingestion rate and exposure frequency. If this information is unavailable, USEPA has provided data from key studies on exposure parameters (USEPA/ORD 1995). It is recommended that the risk assessor use those data that best represent the individual and population behaviors and descriptions for the disposal site. For some exposure parameters, default values are recommended. Any default assumptions that are used may under- or overestimate exposure parameters, adding uncertainty to the overall analysis.

One method for obtaining site-specific information is to use surveys of the local population or creel census data from state fisheries departments or local universities, with review and analysis of the generally accepted survey techniques for the consumption of fish and shellfish.

The USEPA (1992b) does not provide a default value for the fraction of the seafood diet obtained from the management site. Therefore, it will be necessary to estimate this value from site-specific information, fishery statistics, and knowledge of the species in question.

The USEPA does not provide guidance regarding differential consumption of various organs such as muscle, fish skin, fish liver, or other organs, nor is there guidance on other considerations such as food preparation. All of these factors will contribute to the accuracy of the risk estimates and uncertainty in those estimates. Site-specific information may provide insights into local cultural eating habits. In the absence of site-specific information, an assumption may be made for the consumption of finfish: that people consume fish fillets, not the entire fish.

Fraction ingested (FI) for recreational/subsistence scenario. For recreational fishes, in lieu of any site-specific catch statistics, or local information, it should be conservatively assumed that all of the fish consumed by this group is caught within the area influenced by the disposal site. This will represent the most conservative case; although it is likely to be reflective of recreational or subsistence fishermen. Thus, the FI for the recreational/subsistence fishermen would be 1.

FI for commercially consumed scenario. In some areas of the country, individuals purchase seafood from the same vendors who harvest from a particular area consistently. In these cases, the catch is not diluted and the FI would be 1. Typically, commercial catches are not taken from one small area, but from many areas. For commercially harvested seafood, it is best to obtain catch statistics for the area of interest from state departments of marine fisheries statistical reports, or, if necessary, from NMFS statistical reports. Often, the state reports may be on a finer scale, especially for nearshore fishing areas. The species of interest and their foraging areas represented in the statistical areas should be determined by a fisheries biologist. If the state fisheries biologists indicate that the disposal site is particularly attractive to species of concern, then the FI should be adjusted accordingly.

If a site is used repeatedly for dredged material disposal, it may become disproportionately attractive to certain species such as winter flounder because the continual disturbance may enhance populations of opportunistic species. Sometimes these species are the favored prey of winter flounder. State departments of fisheries or local agencies should be consulted regarding this possibility. If it is occurring, the FI should be appropriately modified.

This guidance suggests estimating the FI based on the size of the disposal site relative to the fishery area; the catch from various statistical areas; and the size of the foraging areas for the species of interest.

Example 17: Calculation of FI by Humans Based on Fishery Statistics for Consumption of Commercially Caught Flounder

The State Division of Marine Fisheries' winter flounder catch statistics indicate that 30 percent of all of the flounder landed in the state come from Statistical Area 4. For this example, Area 4 contains the hypothetical dredged material disposal site and its area of influence. It is known that the foraging area of a flounder is approximately 2 percent of Area 4.

Therefore:

$$FI = 0.02 \text{ H} 0.3$$
$$FI = 0.006$$

In this case, the FI for the local metropolitan consumer of commercially harvested flounder is 0.006. Six-tenths percent of the flounder consumed by these receptors will be impacted by the dredge-management site. If there is reason to believe that the disposal site is preferentially attractive to flounder, this calculation will change accordingly.

Reviewing USEPA Default Exposure Assumptions

In the absence of site-specific information for the exposure factors, the risk assessor should use the USEPA recommended default exposure assumptions found in the following four documents.

a. Exposure Factors Handbook (USEPA 1989).

b. Exposure Factors Handbook (USEPA/ORD 1995).

c. Human-Health Evaluation Manual, Supplemental Guidance: Standard Default Exposure Factors (USEPA/OSWER 1991a).

d. Guidance for Assessing Chemical Contaminant Data for use in Fish Advisories. Volume I: Fish Sampling and Analysis (USEPA/OST 1993).

e. Guidance for Assessing Chemical Contaminant Data for use in Fish Advisories. Volume II: Risk Assessment and Fish Consumption Limits (USEPA/OST 1994).

Ingestion rate (IR). Fish consumption rates differ throughout the country, and for specific subpopulations, the use of an "average" consumption rate for all households may not accurately reflect the local consumption rate in a particular subpopulation. It is recommended that the risk assessor review the consumption values presented from key studies identified by the USEPA (e.g., USEPA/ORD 1995). From these data (or others in the literature), exposure factors should be selected.

Absorption fraction (Abs). The absorption of the contaminants from the seafood tissue through the gastrointestinal tract will depend upon the lipophilicity of the compound, the degree to which the lipid soluble portion of the fish is absorbed, and the contents of the human gastrointestinal (GI) tract at the time of ingestion of the contaminated seafood, among other factors.

Exposure frequency (EF). The EF refers to the total number of seafood meals consumed during the exposure duration. This frequency includes seafood harvested from both the dredged material disposal site and elsewhere. This can range from up to 10 meals per year for the recreational fishermen (USEPA/OERR 1992b) to once or twice per week, or more, for those consuming fish harvested commercially or caught by subsistence fishermen (USEPA/Office of Policy, Planning, and Evaluation, and Research and Development 1991). The frequency of consumption of one species can differ from another due to seasonality of catch.

Body weight (BW_{avg}). The default value for average body weight over the exposure period for adults is 70 kg. For children under the age of 6, the default value is 15 kg (USEPA 1989), and for young adults or teens, it is appropriate to use intermediate values.

Exposure duration (ED). The ED represents the length of time over which exposure occurs. Typically, the default values represent upper-bound residential

durations of 30 years and median residential durations of 9 years at a single residence. However, it is recommended that site-specific durations be used. If, for example, the dredged material disposal site will be capped within 2 years of its use, this should be reflected in the exposure duration.

Averaging time (AT). The AT for carcinogenic effects of the contaminants should be 70 years. This is the period (represented in days) over which the exposure is averaged. This is referred to as the Lifetime Average Daily Intake. The averaging time for exposures to noncarcinogens is the exposure duration (in days).

The product of this section is a numerical estimate (a range or single point) of the average daily intake (dose) of a contaminant for each species consumed for each potentially exposed receptor. This information should be presented in tabular format.

Example 18: Intake Calculation for the Consumption of Commercially Harvested Flounder

The risk assessor will calculate a Potential Average Daily Dose of total PCBs due to consumption of winter flounder exposed to the disposal site. The EPC (concentration of total PCBs in the flounder from the area of the site) and FI (fraction of the total catch from the area of the site) have been calculated previously. Note that the EPC is generally expressed as ug/g, although in the intake equation, it is necessary to convert that to mg/kg. The State Department of Marine Fisheries has indicated, in this hypothetical example, that a flounder ingestion rate of 0.11 kg per meal is a conservative estimate of flounder consumption.

$$ADD_{pot}\ (mg/kg/day) = \frac{EPC \times Abs \times IR \times FI \times EF \times ED}{BW_{avg} \times AT}$$

where

EPC = (3.3 ug/g) = 3.3 mg/kg

Abs = 1

IR = 0.11 kg/meal

FI = 0.006

EF = 52 meals/year

ED = 9 years

BW_{avg} = 70 kg

AT = 70 years (365 days/year) = 25,550 days

$$ADD_{pot}(mg/kg/day) = \frac{3.3\ mg/kg \times 0.11\ kg/meal \times 0.006 \times 52\ meals/yr \times 9\ yr}{70\ kg \times 25{,}550\ days}$$

$$ADD_{pot} = 5.6 \times 10^{-7}\ mg/kg/day$$

This is the incremental lifetime average daily intake for the consumption of commercially harvested flounder using conservative, reasonable maximum exposure assumptions.

Toxicity Assessment

This section summarizes the general toxicological information necessary for the completion of the human health risk assessment. The purpose of the toxicity assessment is to provide an estimate of the relationship between the extent of exposure to a contaminant and the likelihood and/or severity of adverse health effects. It considers several types of toxicological information, including human, epidemiological, and animal data. The toxicity profiles provide summaries of the toxicity assessment. Appendix D provides toxicological profiles for the

contaminants of concern likely to occur at dredged material management sites. These should be updated with each risk assessment as indicated in the following steps.

The products of a toxicity assessment are:

a. A discussion of the potential adverse health effects due to exposure to contaminants of concern.

b. The toxicity factors for use in a quantitative estimate of risk.

Step 1: Determine Toxicity Factors

Carcinogenic effects of COC. EPA has used the weight-of-evidence approach to evaluate potential human carcinogens and categorizes them in Integrated Risk Information System (IRIS) (USEPA 1997) and the Health Effects Assessment Summary Tables (HEAST) (USEPA/OSWER 1997). The carcinogenic slope factor (CSF) expresses the carcinogenicity of a compound. The CSF is a toxicity value that defines the quantitative relationship between dose and response. It is a plausible upper-bound estimate of the probability of a response per unit intake of a contaminant over a lifetime. The slope factor is usually the upper 95^{th}-percent confidence limit of the slope of the dose-response curve and is expressed as (mg/kg/day) -1.

Noncarcinogenic Effects of COC. A reference dose, or RfD, is the toxicity value used most often in evaluating noncarcinogenic effects, resulting from exposures to chemicals. The RfD is defined as an estimate of a daily exposure level for the human population, including sensitive subpopulations (such as elderly and children) that is likely to be without an appreciable risk of adverse effects during a lifetime.

Step 2: Assemble Sources of Toxicity Information

There is a hierarchy of toxicity information that should be consulted when conducting a risk assessment. The first is the Integrated Risk Information System (IRIS), an information database that contains chemical-specific health risk and USEPA regulatory information. Information in IRIS supersedes all other sources. If information is unavailable in IRIS, then HEAST may be consulted. The HEAST contains toxicity information and values from USEPA. It is updated quarterly and contains interim toxicity factors that are not found on IRIS.

Human Health Risk Characterization

This text provides the toxicity factors which are quantitative estimates of the potency of the contaminants of concern. These factors, combined with the average daily intake estimates derived in the exposure assessment section, are used to estimate risk in the risk characterization.

Risk Characterization involves the integration of estimates of exposure developed as part of the exposure assessment with health effects information developed as part of the toxicity assessment.

The products of the Risk Characterization section in a human health risk assessment should be:

a. Carcinogenic risk estimates for the reasonable maximum exposed individuals from each pathway, contaminant, and each species of seafood that have been impacted by potential contamination at the dredged material disposal site.

b. Hazard index to evaluate the potential for noncarcinogenic effects from each pathway and COC.

c. A discussion of the risk assessor's confidence in the quantitative estimates.

Carcinogenic risks. The potential for carcinogenic effects is the estimated incremental probability of an individual's developing cancer over a lifetime. This probability is the product of the average daily dose and the CSF. Carcinogenic risk estimates should be calculated by multiplying the chronic (lifetime) average daily intake over a lifetime of exposure by the CSF. Carcinogenic risks should be summed for all pathways for each COC species ingested, unless there is evidence to support segregation of the ingested species.

The equation for estimating incremental lifetime cancer risk (ILCR) for each COC consumed is:

$$\text{ILCR} = \text{Lifetime ADD}_{pot} \text{ H CSF} \qquad (7)$$

The ILCR due to consumption of contaminated seafood impacted by the dredged material disposal site should be estimated by using the lifetime ADD_{pot} that was calculated in the exposure assessment. This should be done for each receptor and species ingested by those receptors.

The total incremental lifetime cancer risk is:

$$\text{Total ILCR} = \sum \text{ILCR}_n \qquad (8)$$

where

$ILCR_n$ = the incremental lifetime cancer risk estimate for the nth seafood species.

Noncarcinogenic effects. The potential for noncarcinogenic health effects is evaluated by the ratio of exposure to toxicity, termed a Hazard Index. The equation for estimating the Hazard Index is:

$$\text{Hazard Index} = ADD_{pot}/RfD \qquad (9)$$

For each exposure scenario, Hazard Indices should be estimated for the consumption of each contaminated species.

Risk Estimates. USEPA (OSWER Directive 9355.0-30, 1991b) states that where the cumulative carcinogenic risk to an individual based on reasonable maximum exposure for both current and future use is less than 10-4 and the noncarcinogenic hazard index is less than 1, further management action is not warranted. The directive also states that site-specific conditions may lead the risk manager to decide that 10-4 is an unacceptable risk based on some site-specific reasons. The risk manager and risk assessor should apply these guidelines when addressing potential human health risk at dredged material management sites.

Example 19: Carcinogenic Risk Estimate for Consumption of Flounder

ILCR = Lifetime Average Daily Intake H CSF

Lifetime Average Daily Intake = 5.6×10^{-7} mg/kg/day
CSF for total PCB = $7.7\ (\text{mg/kg/day})^{-1}$
$ILCR_I$ = 4.3×10^{-6}

EPA generally considers risks in the range of 10^{-6} to 10^{-4} as not indicating a potential human health risk. Therefore, exposure to total PCBs due to the proposed dredging project is unlikely to present a carcinogenic risk to the local human populations. However, this example calculates only risk from exposure to total PCBs. The summed ICLR due to exposure to PCBs and other COCs may present an unacceptable risk.

Note that there is uncertainty associated with this risk estimate because the USEPA currently emphasizes the need for congener specific analyses in assessing risk from PCB exposure.

Uncertainty Analysis

What is Uncertainty Analysis?

Uncertainty analysis is an explicit acknowledgment and analysis of our lack of knowledge of the assumptions and parameters used to assess risk.

How Should the Risk Assessment Address Uncertainty?

The uncertainty analysis should:

a. Identify likely sources of uncertainty in the risk assessment.

b. Identify clearly all significant assumptions at each stage of the assessment.

c. Identify the range and, where possible, the distribution of values which a parameter may take.

d. Test the sensitivity of the risk assessment by using the bounding values for these assumptions (for the most uncertain assumptions).

e. Consider using parameter distributions with a probabilistic technique in the case of large, multipathway risk assessments.

Why pursue an Uncertainty Analysis?

There are three reasons to address uncertainty:

It is a general requirement of most Federal and state risk assessment guidance.

It allows the risk assessor and risk manager to decide whether they have sufficient confidence in the assessment to make a particular management decision.

It allows the risk assessor and risk manager to decide what type of further information they may need to increase the confidence in the assessment.

5 Uncertainty Analysis

Uncertainty is introduced into each step of the risk assessment process. The final risk estimates represent the integration of selected pieces of information, each with its own degree of uncertainty. To address this uncertainty, the risk assessment makes conservative assumptions about potential exposures and toxicity. Therefore, the predicted risk estimates may overestimate actual risks. It is important to recognize that risk estimates are indicators of the potential for adverse effects, not predictors of such effects.

In a risk assessment, there are two ways to describe uncertainty, quantitatively and qualitatively. For most dredged material management activities, uncertainty characterization will typically involve a qualitative discussion of the rationale for using particular scenarios, exposure factors, and data and the level of confidence in those selected parameters. The larger, more complex assessments will require a more quantitative process.

It is possible to express the uncertainty by running the exposure scenarios under various alternative assumptions. These may range from using different statistics for EPCs, varying the frequency of exposure, or changing assumptions regarding the characteristics of the exposure for each scenario. This should be done within the framework of the agreed upon scenarios, and not result in new or separate scenarios involving new receptors, contaminants, or previously unconsidered databases.

The risk assessment should include a qualitative uncertainty characterization that identifies site-related variables and assumptions that contribute to the overall uncertainty in the risk estimates. The uncertainty analysis should:

a. Identify likely sources of uncertainty in the risk assessment.

b. Identify clearly all significant assumptions at each stage of the assessment.

c. Identify the range and, where possible, the distribution of values which a parameter may take.

d. Test the sensitivity of the risk assessment by using the bounding values for these assumptions for the most uncertain assumptions.

Identify Likely Sources of Uncertainty

Obviously, any assumption or measurement introduced into the assessment will have some degree of uncertainty associated with it. In a human health risk assessment, the discussion of uncertainty should address the following assessment elements:

a. The quality and quantity of contaminant concentration in sediment and surface water.

b. The quality and quantity of available data on seafood catch statistics and biota.

c. Use of EPCs in uncooked or whole fish based on modeling of sediment concentrations.

d. Use of surrogate fish species concentration data to estimate average daily intake.

e. Exclusion of dermal and ingestion exposure pathways to the water column.

f. Use of default exposure frequency and duration variables, body weight, life expectancy, and population characteristics.

g. Incomplete understanding of the interaction of contaminants with each other, the mechanism of action of the compounds, and the use of toxicity factors, with their inherent uncertainties such as dose extrapolation and species extrapolation.

The major sources of uncertainty in ecological risk assessment includes:

a. Selection of sensitive ecological receptors.

b. Choice of assessment and measurement end points.

c. Relationship between the assessment and measurement end points.

d. Physical and chemical attributes of the COCS (e.g., partitioning coefficients).

e. Bioaccumulation potential of the COCs.

f. Bioavailabilty of the COCs.

g. Uncertainties in the fate and transport or food chain models.

h. Biological characteristics of the representative species such as foraging range, ingestion rates, migration patterns.

i. Uncertainties in the toxicity factors due to interspecies extrapolations or extrapolating from LOAELs to NOAELs.

Identify Clearly All Significant Assumptions

Significant assumptions are those which the risk assessor feels are most critical to the decision-making process. For example, the selection of a representative species is a critical element because of the underlying assumption that protection of the representative species will afford protection of the ecosystem. Therefore, it is important to be explicit about the importance of this assumption and to present clearly the justification for making it.

Identify the Range Wherever Possible, the Distribution of Values a Parameter May Take

For at least each significant assumption, the risk assessor should provide the range of possible values. For some parameters this information may be available in the literature (e.g., a range of biota to sediment accumulation factors). For other assumptions identifying the range of possibilities may be more difficult. For example, deciding on a "range" of representative receptors is an exercise in professional judgement.

Test the Sensitivity of the Risk Assessment

The risk assessment should include a quantitative evaluation of uncertainty, if possible. Several approaches can be used to characterize uncertainty in parameter values. When uncertainty is high, bounding estimates should be used. Many of the models used in the risk assessment are linear. Therefore, a simple sensitivity analysis should be performed to determine whether the results of the risk analysis are significantly affected by variations within a range (such as BSF or fish ingestion rates).

Sensitivity analysis is the process of changing one variable while leaving the others constant to determine its effect on the output. The results identify those variables that have the greatest effect on exposure and help focus further information-gathering activities; they do not indicate the probability of a variable being at any point within its range. When a single parameter profoundly influences exposure estimates, the assessor may develop a probabilistic description of its range (USEPA/ORD 1995). This can be done using site-specific information (such as creel, market basket, or fish consumption surveys), information in the literature, or data compiled by USEPA.

The most common example of probabilistic uncertainty analysis is the Monte Carlo method. This technique assigns a probability density function to each parameter, then randomly selects values from distributions and inserts them into the exposure equation. Repeated calculations produce a distribution of predicted

values that reflects the overall uncertainty in the inputs to the calculation (USEPA/ORD 1995).

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Appendix A
Summary of Federal, State, and Regional Guidance

There are numerous items which could be included in Appendix A, and we have attempted to include those which will provide the maximum benefit to dredged material disposal problems. We do not intend this section to be a grand "literature review" but rather a presentation and explanation of those risk assessment guidance documents or portions of documents which have some relevance to dredged material disposal problems. Therefore, within this outline, for each item proposed a brief description is provided to define its significance to the overall process.

Appendix A will generally describe the risk assessment process available in the form of Federal, state, and industry group guidance documents.

Federal Guidance

This section describes Federal guidance, summarizes particular requirements, identifies where it is applicable to dredged material disposal issues, and provides information on where to find updates for guidance and guidance support documents

United States Environmental Protection Agency (USEPA) framework for ecological risk assessment

Documents: "Proposed guidelines for ecological risk assessment." (USEPA 1996c).[1] USEPA/630/R-95/002B. USEPA, Risk Assessment Forum, Washington, DC.

"Peer review workshop report on a framework for ecological risk assessment." (USEPA 1992d). EPA/625/3-91/002. USEPA, Risk Assessment Forum, Washington, DC.

Contact: Dorothy E. Patton, Ph.D. Chair, USEPA Risk Assessment Forum. Washington, DC.

[1] A complete list of references is located at the end of the main text.

Significance: These documents, prepared by USEPA Risk Assessment Forum, constitute USEPA's general framework for conducting ecological risk assessments. They provide the broad outlines and general terminology for ecological risk assessment. Most Federal and state guidances borrow from this framework in varying degrees. This subsection summarizes the most recent framework document (USEPA Risk Assessment Forum 1996c) because it includes the principles of the earlier framework document (USEPA Risk Assessment Forum 1992d) and provides the generally accepted state of the practice in terms of the broad goals and methods which an ecological risk assessment should address and apply. This document and associated supporting material provide the basic definitions and processes which comprise the foundation for most Federal, state, and industry sponsored guidance. This summary addresses the questions:

a. What are the major elements, basic definitions, and processes described in this framework?

b. How is the framework being incorporated into current guidance?

c. How is the framework being incorporated in current practice?
e.g. Solomon et al. (1996) assessed risk to surface waters from atrazine. Both studies follow the USEPA three-component model using Problem Formulation, Analysis, and Risk Characterization.

d. What elements of the framework are most adaptable to dredged material disposal problems?

Definitions: This subsection is not a comprehensive risk assessment glossary but provides some fundamental definitions necessary for an informed reading of the framework document and the various guidance documents which follow it.

a. Assessment end point: An explicit expression of the environmental value that is to be protected. An assessment end point includes both an ecological entity and specific attributes of that entity. For example, salmon are a valued ecological entity; reproduction and population maintenance of salmon form an assessment end point.

b. Conceptual model: The conceptual model describes a series of working hypotheses of how the stressor might effect ecological entities. The conceptual model also describes the ecosystem potentially at risk, the relationship between measures of effect and assessment end points, and exposure scenarios.

c. Ecological risk assessment: The process that evaluates the likelihood that adverse ecological effects may occur or are occurring as a result of exposure to one or more stressors.

d. Exposure: The contact or co-occurrence of a stressor with a receptor.

e. Hazard assessment: This term has been used to mean either (1) evaluating the intrinsic effects of a stressor or (2) defining a margin or

safety or quotient by comparing a toxicological effects concentration with an exposure estimate.

f. ***Measure of effect:*** A measurable ecological characteristic that is related to the valued characteristic chosen as the assessment end point.

g. ***Measure of exposure:*** A measurable stressor characteristic that is used to help quantify exposure.

h. ***Receptor:*** The ecological entity exposed to the stressor.

i. ***Risk characterization:*** A phase of ecological risk assessment that integrates the exposure and stressor response profiles to evaluate the likelihood of adverse ecological effects associated with exposure to a stressor. The adversity of effects is discussed, including consideration of the nature and intensity of the effects, the spatial and temporal scales, and the potential for recovery.

j. ***Stressor:*** Any physical, chemical, or biological entity that can induce an adverse response (synonymous with agent).

k. ***Stressor-response profile:*** The product of characterization of ecological effects in the analysis phase of ecological risk assessment. The stressor-response profile summarizes the data on the effects of a stressor and the relationship of the data to the assessment end point.

Summary: "Proposed guidelines for ecological risk assessment" expands upon the earlier EPA framework document, "Framework for ecological risk assessment." Appendix A of the proposed guidelines indicates specific changes that were made from the framework of the EPA's ecological risk assessment. The purpose of an ecological risk assessment, according to this document, is to "organize and analyze data, information, assumptions, and uncertainties in order to evaluate the likelihood of adverse ecological effects." It describes four elements of an ecological risk assessment: planning stage, problem formulation phase, analysis phase, and the risk characterization phase.

The authors of the document also emphasize that an ecological risk assessment is an "iterative" process in which reevaluation and revision is important in each phase.

a. Planning stage. Before beginning the risk assessment, a planning stage, in which there is dialogue between the risk assessor, risk manager, and other parties, should be implemented. The purpose of this planning stage is to ensure that the risk assessment results meet the needs of the risk manager, who is responsible for protecting human health and the environment. In this planning stage, the management goals, scope and complexity, resources needed, and products of the assessment should be discussed and summarized. The document mentions that significant planning is required for a project in which the risk assessment is for a watershed where there are multiple stressors, ecological values, and political factors influencing

the decision-making, such as the Port of New York and New Jersey (NY/NJ) Dredged Material Management Plan (DMMP) project.

The authors place an emphasis on the importance of discussion between risk assessor and risk managers, not only in the planning stages, but throughout the risk assessment process. Consultation between the risk manager and risk assessor is especially important at the beginning and end of the assessment and when the analysis plan is being developed.

b. *Problem formulation phase.* The first phase of an ecological risk assessment, as described in this document, is "Problem Formulation." This phase involves "generating and evaluating preliminary hypotheses about why ecological effects have occurred, or may occur, from human activities." The problem formulation serves as the basis for the rest of the risk assessment. There are basically three products from executing this step:

(1) Assessment end point(s).

(2) A conceptual model.

(3) An analysis plan.

Assessment end points are "explicit expressions of the actual environmental value that is to be protected" (USEPA 1992a). These end points should accurately reflect the ecological concern at the site and focus the risk assessment.

Once these end points are established, a conceptual model of the relationship between stressor(s) and the assessment end points can be developed. The two parts of a conceptual model are a written explanation of the predicted relationships between the stressor and assessment endpoint (risk hypotheses) and a diagram representing the relationships described in the written portion. Justification for the risk hypotheses, as well as uncertainty associated with the proposed conceptual model should be mentioned. An example of a source of uncertainty is if multiple stressors are present at a site. Complex interactions may occur between these stressors which the risk assessor might not predict in a risk hypothesis.

The analysis plan in the "Problem formulation" phase should include the types of data that will be used, the method for data treatment, the assessment design, and level of confidence needed to make management decisions based on available data. Different measures to evaluate risk hypotheses should also be developed, such as measures of effect, measures of exposure, and measures of ecosystem and receptor characteristics. Justifications and uncertainties associated with the analysis plan should also be included.

c. *Analysis phase.* The purpose of the "Analysis phase" of an ecological risk assessment is to evaluate the data that have been collected. The

conceptual model and analysis plan, developed during the "Problem formulation" phase, provide the basis for this "Analysis phase." A characterization of exposure must be conducted during this phase. This characterization can be quantitative or qualitative, depending on the stressor and the scope of the assessment. Exposure is defined as "contact or co-occurrence of stressors with ecological receptors." Exposure can be analyzed by describing "the source and releases of stressors, the spatial and temporal distribution of the stressor in the environment, and the extent and pattern of contact or co-occurrence between the stressor and receptor." Intensity, time, and space are three important factors to consider when estimating exposure. After these factors are taken into consideration in an exposure analysis, an exposure profile should be written to convey the "likelihood that exposure will occur."

Also in the "Analysis Phase," the assessor should identify the ecological effects of interest and conduct an ecological response analysis. The analysis should show how the magnitude of ecological effects changes with varying stressor levels, present evidence that the stressor causes the effect (show "causality"), and link the effects to the assessment end points. Stressor-response relationships can be difficult to assess, especially if multiple stressors are present. However, if the assessor can repeatedly demonstrate cause-effect relationships between the stressor and the effect, then he or she has strong evidence for causality. The document also includes several considerations when linking effects to assessment end points. Judgment approaches, empirical approaches and process-based approaches are presented as general categories of methods to extrapolate effects to assessment end points. The most useful extrapolation approach depends on the parameters outlined in the analysis plan and the conceptual model used. At the end of the ecological response analysis, a stressor-response profile is written to present the results, rationale, and uncertainty of the analysis.

d. *Risk characterization phase.* The third phase of an ecological risk assessment is "risk characterization." The three components of this phase are risk estimation, risk description, and reporting results. The purpose of risk estimation is to "determine the likelihood of adverse effects to assessment end points [identified in the "Problem formulation" phase] by integrating exposure and effects data [from the "Analysis phase"] and evaluating any associated uncertainties." The authors outline the advantages and disadvantages of six approaches for conducting a risk estimate. A risk estimate approach should be chosen based on the amount of data available, the scope of the assessment, and usefulness for risk management. The results of the risk estimate as well as the degree of confidence in the estimate should be included in the risk characterization report.

The goals of the risk description component of this third phase are to make conclusions "about risks to the assessment endpoints," to evaluate the "lines of evidence supporting or refuting the risk estimate(s)," and to interpret the "adverse effects on the assessment end point." Examples of

lines of evidence are quotient estimates, modeling results, field experiments, or field observations. Lines of evidence may be qualitative or quantitative. Five criteria to evaluate if changes in assessment endpoints are "adverse changes" are mentioned in this document. These criteria are:

(1) Nature of effects.

(2) Intensity of effects.

(3) Spatial scale.

(4) Temporal scale.

(5) Potential for recovery.

One hint given for evaluating "adverse effects" is to keep both the ecological and statistical contexts of the results in mind. A complete risk description should also be included in the risk characterization report.

The final section of the "Proposed Guidelines" (USEPA 1996c) is a reminder that the assessor should communicate to the risk manager the major risks to assessment endpoints and the extent of the data supporting the conclusions made in the risk assessment. Then, the risk manager can consider the results of the risk assessment, as well as other social, political, economic, or legal issues to make a decision about further environmental action (if any). The authors of this document also mention that a risk characterization report is a way to communicate ecological risks to the general public. Thus, an ecological risk assessment serves a dual purpose, to guide risk management decisions and to communicate with the public about environmental concerns.

Also in the "Proposed Guidelines" document are several useful appendices. Appendix B defines Key Terms used in ecological risk assessment guidance. Appendices C and D provide examples of conceptual models and analysis phase considerations, respectively. A hypothetical example for evaluating ecological adversity is also given in Appendix E.

Commission on risk assessment

Document: "Risk assessment and risk management in regulatory decision-making." Commission on Risk Assessment and Risk Management, Washington, DC. (Omenn et al. 1996).

Contact: Gilbert S. Omenn (Chairman of the Commission), Dean, School of Public Health and Community Medicine, University of Washington, Seattle.

Significance: This is an important document in that it expresses a clear concern from a Congressionally mandated commission that risk assessment be incorporated into the Federal decision making processes. The United States Congress mandated this commission as part of the Clean Air Act Amendments of

1990 to "make a full investigation of the policy implications and appropriate uses of risk assessment and risk management in regulatory programs under various federal laws to prevent cancer and other chronic human health effects which may result from exposure to hazardous substances."

This document makes several recommendations which have direct implications for the Corps of Engineers DMMP. They are:

a. The Clean Water Act should be amended to establish a comprehensive, integrated watershed-management approach that uses ecological risk assessment and biotic-integrity measurements to provide for the development of state watershed programs.

b. The USEPA and the states should continue to use receiving water quality and risk assessment to set priorities for water pollution control programs, and risk assessment should be used to establish water quality criteria and effluent limits (with the caution that risk assessment, and especially ecological risk assessment, should not yet be used to supplant technology-based and quality-based techniques).

c. The public should be involved in the risk-based decision-making process note that this is consistent with the public coordination process already used in dredged material management.

d. Risk assessment should be in conjunction with a cost benefit analysis as part of the decision making process.

e. Risk assessments should be cautious regarding the use of "bright lines" or numerical criteria.

Summary: This document strongly recommends that risk assessment be incorporated into Federal regulatory decision-making within and among Federal agencies. It emphasizes the involvement of stakeholders in risk assessment and risk management. Also in this report, the Commission presents its vision of a risk management framework, discusses the uses and limitations of risk assessment and of economic analysis, and makes specific recommendations for the use of risk assessment in Federal regulatory agencies and programs.

The philosophy of the Commission regarding risk management and risk assessment is that the problem or concern should be formulated in a broad context. They comment that risk analysis is often based on the effects of individual chemicals on human or environmental receptors. The Commission calls for the risk assessor to consider how mixtures of chemicals may act in various media to cause "chronic health effects." They also state that the focus of risk analysis should be to protect public health and the environment by considering realistic scenarios and scientific methods.

The report also discusses three risk assessment issues currently under debate:

a. One of the issues discussed is the assessment of toxicity and relevance to humans. The Commission suggests that a common metric is needed to

compare the risk of cancer and noncancer effects, such as "margin-of-exposure" ratio which is currently used in a limited capacity by the USEPA.

b. A second issue of concern is how risk assessments account for variations in population exposure and susceptibility. The Commission recommends distributional approaches (i.e., probabilistic risk assessment) to more accurately address variations in exposure and susceptibility.

c. A third issue under debate is describing uncertainties associated with risk assessments. The Commission highlights the importance of communicating both quantitative and qualitative risk to risk managers. The importance of focusing risk assessment on other forms of risk, such as that associated with microorganisms and radiation, in addition to risk posed by chemicals is also expressed.

The Commission proposes the involvement of stakeholders in the ecological risk assessment process, as well as in human health risk assessment. It notes that the current framework for conducting ecological risk assessments needs supplemental guidance regarding the involvement of stakeholders in ecological risk assessment.

The next section of the report addresses the use of cost analysis in conjunction with risk assessment in regulatory decision-making. The Commission states that, "Considering costs and benefits in regulatory decision-making can help to clarify the tradeoffs and implications associated with alternative regulatory policies and help regulatory agencies to set priorities." Two forms of cost analysis are highlighted in this document. One is "cost-effectiveness analysis" which can help choose an option which meets a specific regulatory goal for the least amount of money. The second form of cost analysis is "benefit-cost analysis" which is used to "assess the benefits and cost of different health-based standards with *different* levels of health protection."

There is also a section of the Commission's report which discusses communication and comparison of risk. The Commission stresses the fact that risk assessors, risk managers, stakeholders, and the public all have different perceptions of risk. However, risk assessment can help to reach a consensus regarding priorities for environmental health and safety ("comparative-risk ranking"). Also in their report, the Commission cautions risk assessors in the use of "bright lines," "numerical values between unacceptable and negligible magnitudes of risk or exposure concentrations of concern." "Bright lines" should be used as goals for decision-making but should not be applied inflexibly. The Commission further expresses its view on the importance of peer review in risk assessment and that laws expanding judicial review to cases regarding agency compliance with "detailed procedural requirements" or "the resolution of complex scientific issues" should not be supported.

The next section of the document outlines current Federal agency risk assessment and risk management practices. Also, within the USEPA, recommendations are made for the incorporation of risk assessment methods to

the Office of Air and Radiation, Superfund, Office of Prevention, Pesticides, and Toxic Substances, and the Office of Water. Recommendations are also made to Occupational Safety and Health Administration (OSHA), the Food and Drug Administration (FDA), Department of Agriculture, Department of Energy, and Department of Defense. Currently, the Superfund Program, created by Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA) and administered by the Office of Emergency and Remedial Response (OERR), has made use of risk assessment to a greater extent than other agencies. Superfund uses risk assessment to define hazardous substances and amounts of release that must be reported to the USEPA, rank risks posed by hazardous waste sites and identify "action priorities" among sites, and evaluate the effectiveness of options for remediation. "An important and unique feature of Superfund risk assessments is the consideration of exposure to many chemicals simultaneously." Specific policies on risk assessment are in the National Contingency Plan (NCP), the body of regulations implementing CERCLA and its amendments. Risk analysis is also currently used for regulatory decision-making under six major environmental laws and a number of minor laws.

USEPA Environmental Response Team

Document: "Ecological risk assessment guidance for superfund: Process for designing and conducting ecological risk assessments." United States Environmental Protection Agency Environmental Response Team, Edison, NJ. (USEPA 1997a).

Contact: David Charters, Ph.D., USEPA Environmental Response Team, Edison, NJ.

Significance: Unlike the USEPA framework documents, this guidance is a step-by-step procedure for assessing ecological risk at CERCLA sites nation wide. Many USEPA regional guidance documents and state documents borrow from the procedures in this document and its earlier 1994 version. Much of the detail in this document is specific to superfund sites and therefore not directly transferable to the dredged material management process. However, the techniques approaches for developing conceptual models, using a screening analysis step, and developing Scientific management decision points are useful in the dredged material management process.

Summary: In this document, the USEPA provides guidance for conducting scientifically sound ecological risk assessments that are consistent with other risk assessments within the Superfund Program. This guidance is directed to site managers (e.g., On-Scene Coordinators (OSCs) and Remedial Project Managers (RPMs)) as well as other parties conducting ecological risk assessments. The goals of an ecological risk assessment, as stated in this document, are to:

> "identify and characterize the current and potential threats to the environment from hazardous substances" and "to identify clean-up levels that would protect those natural resources from risk."

This document describes, with examples, eight steps for doing an ecological risk assessment. Steps 1 and 2 of the EPA guidance are designed to allow risk assessors and risk managers to quickly determine whether a site poses a risk to the environment.

Step 1 - Screening-level problem formulation and ecological effects evaluation

In the problem formulation component of Step 1, all parties involved in the risk assessment, including site managers, risk assessors, the Biological Technical Assistance Group (BTAG), the Potentially Responsible Party (PRP), and stakeholders, work together to define the goals of the assessment and to propose a scope for remedial action. This problem formulation step is critical to focus the scope of work for the risk assessment. Step 1 also includes a toxicity evaluation to determine what specific component of the ecosystem could be adversely affected by contaminants from the site (ecological effects evaluation).

Step 2 - Screening-level estimate and risk calculation

The goal of this step is to decide whether a significant ecological risk has been identified, based on screening assessment results, or if a more detailed risk assessment should be conducted. At the end of Step 2 is a scientific/management decision point (SMDP). SMDPs occur at defined points in the assessment process. The purpose of SMDPs is to guide work, discuss the uncertainty of risk assessment, and to keep lines of communication open between parties working on the assessment. In this way, SMDPs ensure that time and money are not wasted due to flawed decisions, miscommunication, or misunderstandings while conducting the risk assessment.

Step 3 - Baseline risk assessment and problem formulation

If the results of the screening-level assessment from Steps 1 and 2 prove insufficient to rule out risk to the environment or if they indicate that some significant risk is present, then the assessment proceeds to Step 3. This step uses the screening assessment results in conjunction with more site-specific information to refine the problem formulation and expand on ecological issues of concern. Specifically, assessment endpoints, exposure pathways, and risk questions are developed. Step 3 also involves the development of a site conceptual model, which integrates the above three components. The purpose of the SMDP at the end of Step 3 is to determine if this conceptual model is acceptable.

Step 4 - Study design and data quality objective process

Step 4 uses the conceptual model developed in Step 3 to define measurement endpoints, data quality objectives (DQOs), and the study design. These components are directly integrated into the products of Step 4, an ecological risk assessment work plan (WP) and a sampling and analysis plan (SAP). The WP and SAP are critical to be able to gather enough information for the risk assessor to

make a good prediction of risk. This document outlines what the basic contents of a WP and an SAP should be and that they should follow the EPA model for work plans and SAPs. The end of Step 4 also has an SMDP to approve the work plan and sampling and analysis plan that will be used in the next steps of the risk assessment.

Step 5 - Field verification of sampling design

In this step, the information collected thus far is verified and the feasibility of sampling is evaluated after visiting the site. Some elements of the WP or SAP may need to be modified to meet the objectives of the risk assessment. These changes can be made in consultation with the risk assessor and risk manager. If a reevaluation of the assessment endpoints in the WP or SAP is needed, however, the assessor must return to Step 3. The SMDP for Step 5 is the signing of the finalized WP and SAP.

Step 6 - Site investigation and analysis phase

This step involves field sampling and surveys as well as analysis of exposure and ecological effects. Field sampling implements the plans designed in Step 5 to collect data. The analytic approach for characterizing exposure effects and ecological effects is outlined in the WP and SAP and are based on the site conceptual model. After sample collection and analysis of exposure and ecological effects, an exposure-response analysis is conducted. This analysis relates the "magnitude, frequency, or duration of contaminant stressors ...to the magnitude of the response." Also, "measurement endpoints [measures of effects] are related to the assessment endpoints using the logical structure provided by the conceptual model." An SMDP is needed in Step 6 only if the WP or SAP needs to be altered.

Step 7 - Risk characterization

The risk characterization is a "qualitative and quantitative presentation of risk and the associated uncertainties." The risk characterization involves risk estimation and risk description. In the risk estimation component, the assessor must describe the methods used and reasoning behind the connections made between exposure profiles and exposure-effects information. The risk description provides information important for interpreting risk results and identifies a threshold for adverse effects on assessment endpoints. One caution that the guidance makes is to clearly distinguish between uncertainty and variability in the description of risk.

Step 8 - Risk management

This step is the responsibility of the site risk manager. There must be a balance of the risk reduction associated with cleanup with the potential ecological impacts of the cleanup process itself. The decision must be made whether or not to clean

up the site to within the range set in Step 7. A risk manager must take many factors into consideration when making a decision. These factors include:

a. Compliance with regulations.

b. Long-term and short-term effectiveness.

c. Cost, state acceptance.

d. Community acceptance.

At the end of Step 8 is an SMDP for approval of the risk management decision, which is finalized in a Record of Decision (ROD).

This guidance document clearly outlines the necessary steps to conduct an ecological risk assessment. It also includes a glossary of important terms, and appendix entitled "Example Ecological Risk Assessments," an appendix entitled "Supplemental Guidance on Literature Search," an appendix on "Statistical Considerations," and a copy of the "Representative Sampling Guidance Document, Volume 3: Ecological" (USEPA/ERT 1997).

USEPA CERCLA Guidance Documents

Document: "Risk assessment guidance for superfund, Volume I, Human-health evaluation manual (Part A), Interim final," (USEPA/OERR 1989a).

Document: "Risk assessment guidance for superfund, (RAGS) Volume II, Environmental evaluation manual, Interim final" (USEPA/OERR 1989b).

Document: "Risk assessment guidance for superfund, Volume I, Human health evaluation manual (Part C) Risk evaluation of remedial alternatives," (USEPA/OERR 1991).

Document: "Guidance for data usability in risk assessment (Part A), Final publication" (USEPA/OERR 1992b).

Document: "Guidance for the data quality objectives process" (EPA/600/R-96/055), USEPA Quality Assurance Management Staff, Washington, DC. (USEPA/ORD 1994).

USACE Guidance Documents

Document: "Risk assessment handbook human health evaluation," EM 200-1-4, U.S. Army Corps of Engineers, Washington, DC. (Headquarters, USACE 1995).

Contact: None Given

Significance: The significance of this handbook lies in the fact that it is a USACE Engineer Manual which provides guidance for conducting human health risk assessments at CERCLA and (Resource Conservation and Recovery Act (RCRA) sites. The document stresses adherence to EPA risk assessment guidance and also describes the importance of integrating the risk assessment into a larger risk management framework.

Summary: This handbook "provides the minimum requirements for developing scopes of work, evaluating Architect-Engineer (A-E) prepared human health risk assessments, and documenting risk management options for risk assessors." The guidelines presented in this document are consistent with and should be considered in addition to "Risk assessment guidance for superfund, Vol. I: Human health," (USEPA OERR 1989a) and "Data usability for risk assessments," (USEPA OERR 1992b). Also, the focus of the document is human health evaluations for Superfund sites (under CERCLA) and RCRA sites (see Glossary for definition of acronyms). The USACE also applies Department of Defense (DOD) policies in their human health evaluations.

CERCLA and RCRA integrate risk assessment into hazardous, toxic, and radioactive waste (HTRW) investigations. The basic components of a human health risk assessment at superfund sites are data collection and evaluation, exposure assessment, toxicity assessment, and risk characterization. Under RCRA, the EPA defers protection of health of onsite workers to OSHA, but a customer may request an assessment of short-term and long-term risks associated with a RCRA site. The authors of this document state that HTRW risk assessments should present a range of exposures to human receptors, and not assess risk solely based on the "worst case" or the "most exposed individual" (MEI).

There are four phases of the HTRW data quality design process used to develop a scope of work for a risk assessment.

a. Phase I is the development of a site strategy, which includes "customer communication of needs and understanding the regulatory requirements/ basis for making site decisions" and the involvement of appropriate project personnel.

b. In Phase II of the data quality design process, data needs are determined. The output from Phase II is a scope of work and a description of these data needs.

c. Phase III is where data collection options are identified, assembled, and presented.

d. In Phase IV, where the data collection program is selected DQOs are assigned. Uncertainties, cost/benefits, and a schedule associated with data collection are presented, as well.

CERCLA and RCRA are functionally equivalent in regard to risk assessment requirements. The project phases for a site investigation are similar and the

decisions made at each phase are similar. The USACE describes four phases in the site investigation process.

a. Phase I is a preliminary risk screening of the site (known as PA/SI under CERCLA and an RFA under RCRA).

b. Phase II, a baseline risk assessment (BRA), is performed in an Remedial Investigation (RI) or Remedial Feasibility Investigation (RFI).

c. Phase III is a risk-based analysis of remedial alternatives in which different options are evaluated for their potential to reduce the baseline onsite risk Feasibility Study (FS) or Corrective Measures Study (CMS).

d. In phase IV, the short-term risks associated with remediation of a site are assessed in a Resource Damages/Risk Assessment (RD/RA), CMI, removal action, or interim corrective measure.

Within each phase of the site investigation process, the USACE defines five steps for determining data needs.

a. *Step 1.* Background information review, site features, hazard information, and exposure information are collected.

b. *Step 2.* Using the information collected in Step 1, a project decision statement (PDS) is made which states whether the assessment should continue or whether the site can be eliminated from concern.

c. *Step 3.* The data requires identification defining project study elements. This step includes the development of a site conceptual exposure model (SCEM). Note: In phase III of site investigation, two SCEMs are needed. One SCEM is for the site during remediation or implementation of corrective measures, and the other SCEM is for the site after remediation.

d. *Step 4.* The risk assessor must define and group data needs and describe the methodology used to analyze the data.

e. *Step 5.* The data needs must be documented.

The authors of this document emphasize that risk assessments should consider risk management needs. For example, "Under the PA/SI or RFA phase, screening risk assessment and exposure analysis may be performed to determine the need for further investigation." In Phase II (Remedial Investigation (RI)), the results of a BRA are used to develop cleanup levels during the next phase (Feasibility Study (FS) or Corrective Measures Study (CMS) phase). The purpose of an FS or CMS is to provide a quantitative and qualitative evaluation of potential health impact from remedial alternatives. Two types of risk assessments are done in an FS or CMS. One assessment is done to develop chemical-specific remediation goals (RGs) to be applied to site cleanup. The purpose of the other assessment is to evaluate the short and long-term risk associated with each alternative.

Risk assessments can be used by risk managers to prioritize or sequence remedial work performed. This document presents risk management options and requirements for action. In deciding what action to take at a site, the risk manager must consider the risk information needed, the risk information provided by the risk assessment, products or deliverables, and risk management options/rationale. There are also several issues, unrelated to risk, that influence risk management. These nonrisk issues include available and usable technology for cleanup, duration of the project, data uncertainty, enforcement, compliance, schedule, budget, compliance with Federal and state laws, community input, and societal and economic value of the resources to be protected. It should be noted that "the NCP recognizes that it is not possible to achieve zero risk in environmental cleanup; therefore, the approach taken by Superfund is to accept non-zero risk and return the site to its best current use, not use in the pre-industrial era." All of the above factors affect the use of risk assessment data by risk managers in HTRW investigations.

Document: Puget sound dredged disposal analysis reports – "Framework for comparative risk analysis of dredged material disposal options, Seattle District," (Tetra Tech 1986).

Document: "Guidance for conducting risk assessments at United States Army sites (Wentsel et al. 1994).

Regional Guidance

This section reviews risk assessment guidance developed by USEPA Regions. In particular, it will assess the relationship between regional and state guidance and its application (if any) to related regulatory programs such as water-quality certification and coastal zone consistency. Many regions have separate guidance for conducting risk assessment which may impose distinct requirements on the performance of risk analysis process and which incorporate changes in approach not yet adopted by national guidance.

State Guidance

This section reviews human health and ecological risk assessment guidance developed by various states. The states often integrate a tiered approach to risk assessment which is amenable for use in evaluating risks at dredged material management sites. These tiered approaches variously use water-quality criteria, sediment screening levels or effects levels, and area of contamination in a tiered approach to risk assessment. Included in this section are the coastal and inland states bordering major water bodies (e.g. Mississippi; Great Lakes).

Note that these guidance documents are generally directed toward conducting risk assessments at RCRA or state hazardous waste sites. As such, not all aspects of these state guidance documents will apply to dredged material management activities, but the general principles will apply.

Alaska

The Alaska Department of Environmental Conservation, Contaminated Sites and Remediation, is currently working on a guidance document that addresses human health and ecological risk assessment. Public comments on the draft of the document have been received and a second draft of the document was due in December 1997. The draft of the document is currently available on the Internet at:

www.state.ak.us/local/akpages/env.conserv/dspar/csites/tp.htm

California

Document: "Guidance for ecological risk assessment at hazardous waste sites and permitted facilities, Part A: Overview and Part B: Scoping assessment," (CA DTSC 1996).

Summary: Part A - The Human and Ecological Risk Division, Department of Toxic Substances Control (DTSC) within the California Environmental Protection Agency wrote this guidance. It is a tiered approach to ecological risk assessment and provides a framework and conceptual model for assessing impact of chemicals to biota. The document also provides guidance for estimating threshold concentrations for each chemical of concern and for incorporating the findings from the ecological risk assessment in remedial, permitting, or control actions.

There are three phases in this risk assessment framework.

a. Phase I is a scoping assessment to predict potential chemicals and receptors of concern and exposure pathways, develop a site conceptual model, and identify any further work that is needed. The authors assume in their risk assessment that individual or population level effects have an impact at higher levels (e.g. communities or ecosystems), unless there is evidence to the contrary.

b. Phase II of the assessment is a "validation study" which involves sampling and analysis of data to refine the Phase I assessment of risk to biota. If the assessor decides to remediate the site at this point, the data gathered in Phase II can be used to develop remediation goals.

c. A Phase III "impact assessment" further investigates the risk to biota at a particular site. In this step, the severity and extent of ecological impact(s), including remediation impacts, are considered. This information is used to help the risk manager decide upon a remediation alternative.

Part B - Part B of California's state guidance for ecological risk assessment explains the Phase I scoping assessment in detail. The purpose of Phase I is to identify potential receptors, chemicals of concern, and complete exposure pathways. A list of chemicals of concern can be developed using the site-specific history of use of the site or using laboratory testing of media. The former is more commonly used in the Phase I assessment. The assessor must justify the inclusion or exclusion of chemicals of concern in the risk assessment and consider the

differences between chemical toxicity to biota and to humans. At this point, if no organic chemicals of concern are present or the concentration of inorganic elements is at or below "background," the site does not need to be assessed further.

If the assessor decides to continue the evaluation, a biological characterization of the site is conducted. The assessor visits the site and identifies habitats and "special species" of concern. Next, exposure pathways are identified. The authors use a "habitat approach as a basis for identifying potential exposure pathways between the areas of contamination and specific species or habitats which occupy, or potentially could occupy, the site." The authors suggest that contact between biota and COCs in media such as soil, air, water, and biota, and by direct and indirect routes be considered.

The product of this assessment is a qualitative evaluation of the threat to nonhuman receptors. The authors suggest that a qualitative statement of the magnitude, duration, and frequency of exposure to receptors to each contaminant or area of contamination be included in the risk assessment report. The minimum requirements for figures, tables, and data are also given in this document. At the end of Phase I, the assessor must also submit a Work Plan Outline to the DTSC. The required contents of this outline are also specified in this guidance document.

Document: "Supplemental guidance for human health multimedia risk assessment of hazardous waste sites and permitted facilities" (California EPA/DTSC 1996).

The California DTSC has a guidance manual on human health risk assessment which supplements the USEPA's "Risk assessment guidance for superfund, Volume I, Human health evaluation manual (Part A) and USEPA Office of Solid Waste and Emergency Response directives. This manual provides recommendations on specific technical or scientific issues that may be encountered when preparing human health risk assessments. The document generally follows USEPA guidance and provides specific information on:

a. Default Exposure Parameters.

b. Use of soil concentration data in exposure assessment.

c. Selection, use, and limitations of indicator chemicals for evaluation of exposure to complex waste mixtures.

d. Assessment of health risks from inorganic lead in soil.

e. DDT in soil: Guidance for the assessment of health risk to humans

f. A toxicity equivalency factor procedure for estimating 2,3,7,8-tetrachlorodibenzo-p-dioxin equivalents in mixtures of polychlorinated dibenzofurans.

Florida

Document: "Guidelines for the preparation of contamination assessment reports for petroleum contaminated sites." (Department of Environmental Regulation, State of Florida Bureau of Waste Cleanup Technical Review Section 1989).

The Florida Department of Environmental Protection has prepared this document. It is driven by human health considerations rather than ecological considerations. The state does not have a formal ecological risk assessment guidance document.

Georgia

Document: "Guidance for selecting media remediation levels at RCRA solid waste management units." Georgia Environmental Protection Division, Atlanta (1996).

Summary: This document outlines the use of risk assessment to determine remediation levels at RCRA facilities. The determination of risk-based remediation levels fits into the RCRA Facility Investigation (RFI) step in the RCRA Corrective Action Progress. The purpose of the RFI is to characterize the nature and extent of releases, assess the risk posed by those releases, and identify potential media remediation levels. This document presents general requirements for using risk assessment to determine remediation levels that are protective of human health and of ecological receptors.

To assess risk to human receptors, chemicals of potential concern (COPCs) for each medium must be identified. If the risk or hazard of a particular COPC exceeds certain "trigger levels," then the chemical is designated a chemical of concern (COC). The "trigger levels" are:

a. *Carcinogenic risk* - if the cumulative cancer risk ("the summed risk to a receptor of all COPCs for all pathways per land use scenario") is greater than 1×10^{-6}.

b. *Noncarcinogenic risk* - if the total hazard quotients (HQs) of all COPCs for all pathways per land use scenario (HI) exceed 1.

Remediation levels for the protection of human health are backcalculated using Region IV Supplemental Guidance to RAGS attached to this guidance document for each COC (not COPC) in each affected medium. A justification of each remediated value must accompany the value in the RFI report.

Ecological risk assessment under the Georgia guidance employs a screening approach. The initial step is a preliminary risk evaluation (PRE). The PRE uses a benchmark approach RCRA facilities in which the facility-related contaminant concentrations are compared with USEPA Region 4 ecological screening values. If the screening values are exceeded, four steps are followed:

1. Problem formulation.
2. Ecological effects evaluation.

3. Exposure estimation.

4. Risk calculation (using the HQ method to calculate a Hazard Index (HI)).

If the HI (sum of the hazard quotient for all COCs) exceeds 1, an ecological risk assessment (ERA) must then be conducted.

The Georgia Environmental Protection Division (EPD) suggests that the assessor at the facility consult with them to determine appropriate assessment and measurement end points. An HQ or an HI is calculated in the ERA using site-specific data, and proposed remedial levels are developed for each COC in each medium for each receptor whose HQ exceeds 1. As in the development of remediation levels which are protective of human health, remediation levels are backcalculated and must be justified in the risk assessment report.

The remediation levels which are protective of human receptors and those which are protective of ecological receptors are compared and the lesser of the two values is selected as the final remediation level. Final remediation levels must be protective of human and ecological receptors, as well as protective of groundwater quality. Further, remediation must achieve protective levels for current and "reasonable anticipated future" uses of the facility. The Georgia Environmental Protection Division must approve all risk-based remediation levels.

Also in Georgia, the Department of Environmental Quality has started a risk-based program in their Hazardous Waste Division for determining risk to human health. No guidance document exists at this time, but a document that models Agency for Toxic Substances and Disease Registry (ATSDR) guidelines may be written in the future.

Louisiana

The Louisiana Department of Environmental Quality (DEQ) currently is working on a document entitled Proposed Approach for Implementing a Louisiana Department of Environmental Quality Risk-Based Corrective Action (RBCA) Program. The period for public comments on the document has just ended (June 1997) and the document will now be revised. This RBCA program is intended to be used by all of the DEQs programs, including the Hazardous Waste Division, Solid Waste Engineering Division, and Water Quality Management Division.

Maine

Document: "Guidance manual for human health risk assessments at hazardous substances sites. State of Maine Department of Environmental Protection and the Department of Human Services (ME DEP 1994).

This document was developed by the State of Maine Department of Environmental Protection and the Department of Human Services in June 1994. Its purpose is to provide consistent and "scientifically sound" procedures for conducting human health risk assessments. It provides guidance for conducting a

baseline risk assessment that does not consider site-specific conditions during or after remediation. It is consistent with the USEPA's CERCLA guidance, and also incorporates some components specific to Maine. For example, it provides default exposure factors for residents of Maine which will be useful in developing human health exposure assessments and risk characterizations for dredged material management activities in Maine.

This manual has eight sections, each corresponding to a separate step in the risk assessment process.

a. Section 1 describes the preliminary steps for a risk assessment, which includes visiting the site and defining the study area.

b. Section 2 describes how to conduct a hazard identification. Hazard identification involves developing or reviewing a sampling plan and analytical methods, as well as collecting, analyzing, and summarizing data.

c. Section 3 is an exposure assessment. To conduct an exposure assessment, the assessor must construct exposure scenarios, estimate exposure point concentrations, and estimate doses of the contaminants to "populations of concern."

d. Section 4 is a dose-response assessment which integrates the toxicity of specific contaminants with the exposure scenarios for a specific receptor coming into contact with those contaminants. In the dose-response assessment, both noncarcinogenic and carcinogenic effects must be considered. Exposure is considered to be chronic (>7 years) in this assessment, as well.

e. Section 5 describes risk characterization using the incremental lifetime cancer risk (for carcinogenic compounds) and the Hazard Index (for noncarcinogenic compounds).

f. Section 6 requires a risk assessment for subchronic (2 weeks to 7 years) and acute (2 weeks) exposures.

g. Section 7 is an analysis of uncertainty.

h. Section 8 requires the risk assessment to prepare the selection of cleanup target concentrations. The following components must be included in this step: "backcalculations" of factors to derive target exposure point concentrations, an evaluation of the effects of leaching to groundwater, and a comparison of the concentrations of chemicals on the site to regulatory standards and guidelines.

In addition to the eight sections of the document, there are some appendices which provide more details for conducting a human health risk assessment. The appendices explain procedures for developing a cancer potency factor and reference dose for deriving exposure factors and target exposure point

concentrations and for establishing the effects of subchronic and acute exposures. The format requirements for the risk assessment report are also given.

Massachusetts

Documents: "Massachusetts contingency plan," 310 CMR 40.000 (MCP 1993).

"Guidance for disposal site risk characterization." Massachusetts Department Environmental Protection (BWS/ORS-95-141).

Contact: Mr. Paul Locke, Massachusetts DEP, Office of Research and Standards, 1 Winter Street, Boston, MA.

The Massachusetts Contingency Plan (MCP) provides a tiered approach which uses three methods for human-health risk characterization. Each method becomes more site specific.

1. In a Method 1 risk assessment, benchmark values or standards for chemicals of concern in soil and groundwater are used as conservative estimates to assess risk.

2. In a Method 2 assessment, the assessor may derive new method standards for compounds for which the MCP does not have standards and/or may modify existing standards based on site-specific fate and transport information.

3. In a Method 3 risk assessment, site-specific exposure assumptions are used to characterize potential risks.

In the cases of Methods 1 and 2 assessments, "a condition of no significant risk of harm to health exists if no Exposure Point Concentration is greater than the applicable standard." For a Method 3 assessment, however, the cumulative cancer risks and cumulative noncancer risks are calculated and compared to the cumulative carcinogenic or noncarcinogenic risk limit, respectively. The basic steps of a risk characterization are: identify human receptors; identify environmental receptors; identify site activities and uses; identify exposure points; identify exposure pathways; identify exposure point concentrations; identify site groundwater and soil categories.

Ecological risk assessments under the MCP have a two-stage approach. A Stage I environmental screening eliminates pathways from Stage II consideration if:

a. Significant risk is readily apparent.

b. Exposure pathway is incomplete.

c. Pathway is incomplete, but the exposure is so minimal that it clearly does not pose a significant risk.

The Massachusetts guidance is of interest to dredged material disposal activities because it explicitly defines background and allows a consideration of local conditions in eliminating contaminants of concern from the risk assessment.

a. Background concentrations are those which are ubiquitous and consistently present near the disposal site and are attributable to geological or ecological conditions, atmospheric deposition of industrial or engine emissions, fill materials containing wood or coal ash, releases to groundwater from a public water supply system, and/or petroleum residues that are incidental to the normal operation of motor vehicles. Compounds which are consistent with background are not assessed further.

b. Comparison to local conditions is another step which the guidance recommends only for aquatic environments, specifically sediment and surface water. Local conditions are levels of oil and/or hazardous material present consistently and uniformly throughout the surface water body, or throughout a larger section of a river that contains the area potentially affected by contamination at or from the site. Hot spots and localized contamination are not considered local conditions. Like background, local conditions may be assessed on a chemical-specific basis. When concentrations are consistent with local conditions, further assessment of the risk posed by that substance in that medium may not be required.

In aquatic environments, the detection of elevated levels of contamination in sediment or surface water, or the potential for elevated levels to occur in the future constitutes identification of a complete exposure pathway. For any complete pathway, effects-based screening is necessary in Stage I. For effects-based screening levels, the guidance recommends National Oceanic and Atmospheric Administration (NOAA) Effect Range – Low) ER-Ls (Long et al. 1995) for marine and estuarine sediment, Ontario Ministry of the Environment Guidelines for freshwater sediment (Persuad 1993), and Ambient Water Quality Criteria (AWQC) or Lowest Observed Effect Level (LOELs) (in that order) published for surface water. For a Stage I screening for wetlands, again the complete exposure pathways should be identified and then an effects- based screening should be conducted. Note that the effects screening criteria are only for ruling out pathways, not individual chemicals. If a pathway is not ruled out, risk from all chemicals that result in exposure by that pathway should be evaluated in Stage II, even if those substances are present at levels below their screening criteria.

The Stage II ecological risk assessment follows the general framework of USEPA ecological risk assessment guidance. The process includes problem formulation, analysis of exposures and associated ecological effects, and risk characterization, which integrates exposure and effects analysis. Also, an uncertainty analysis should be included in the risk characterization. The risk of harm to the environment is characterized by comparing the concentration of each oil or hazardous material to the upper concentration limits in soil and groundwater.

New Hampshire

Document: "Contaminated sites risk characterization and management policy" (September 1996).

Contact: New Hampshire Department of Environmental Services, Concord, NH.

Summary: The New Hampshire Department of Environmental Services' guidance document describes "a tiered risk-based approach to characterize risks to human health and environment posed by the release of contaminants at sites in New Hampshire." The State's guidance for management of contaminated sites borrows directly from Massachusetts regulation in its approach to assessing risk in surface water bodies. It also follows the general format and tiered approach (Methods 1, 2, 3) for risk assessment in the Massachusetts Contingency Plan.

New York

Document: "Technical guidance for screening contaminated sediments" (New York State Department of Environmental Conservation 1999).

Contact: Albany, NY.

This document contains guidance for identifying areas of sediment contamination and making a preliminary assessment of risk to human health and the environment. Guidance for deriving criteria for nonpolar organic contamination and metals in sediment is the focus of the document. Sediment criteria for nonpolar organic contamination are derived using the equilibrium partitioning approach, which assesses biological impact based on affinity of a chemical to sorb to organic carbon in the sediment. Contaminant-specific New York State water-quality criteria for protection of human health and piscivorous wildlife are also used to derive sediment criteria for nonpolar organic contamination. USEPA ambient water quality criteria were used when state water quality criteria for a specific contaminant were not available. Sediment criteria for metals are derived from effects-based concentrations, such as the Ministry of Ontario Guidelines for Protection and Management of Aquatic Sediment Quality and the National Oceanic and Atmospheric Agency (NOAA) ER-Ls and ER-Ms. The lowest concentration from either of these effects-based concentrations (Ontario or NOAA) is selected as the sediment criterion.

The concentration of nonpolar organic contamination and metals in the sediment at a site are compared to the screening criteria concentrations described above. If the sediment criteria are exceeded, a site-specific evaluation of the contaminated sediment must be conducted. Further evaluation generally includes additional chemical testing, sediment toxicity testing, and sediment bioaccumulation tests. Technical guidance for conducting a site-specific evaluation is not given in this document.

The ultimate goal of this screening process for contaminated sediment is to make a decision regarding remediation of the site. Several factors such as the volume and location of the sediment exceeding a sediment criterion, persistence

of the contaminant, and uncertainty of the criteria must be considered as part of deciding upon a remediation action for the site.

Document: "Fish and wildlife impact analysis for inactive hazardous waste sites" (New York State Department of Environmental Conservation 1994). Albany, NY.

North Carolina

Guidance in Development

Oregon

Document: "Guidance for ecological risk assessment: Level I - Scoping" (Oregon DEQ 1997a).

Contact: Oregon Department of Environmental Quality.

The Oregon Department of Environmental Quality's ecological risk assessment process consists of four levels (or tiers):

1. Level I – Scoping.
2. Level II – Screening.
3. Level III – Baseline.
4. Level IV - Field Baseline.

The guidance document for the Level I assessment was completed in April 1997, and a draft of guidance for Level II is currently available. Guidance for Levels III and IV have not been written at this time.

The purpose of a Level I ecological risk assessment is to make a qualitative determination of whether a release or suspected release of a hazardous substance poses a potential risk to ecological receptors. The first task that must be completed in a Level I scoping assessment is to assess/gather existing data about the site. Then, the assessor or an ecologist or biologist with risk assessment experience must make an initial site visit. After these two tasks are completed, the next step is to identify contaminants of interest (COIs) at the site. This is generally done using site-specific historical information at this level. Using the information gathered in the previous steps, the assessor can then evaluate receptor-pathway interactions by considering whether complete pathways for exposure of important species or habitats to the COIs are present. Complete exposure pathways are defined as those that have:

> "a source and mechanism for hazardous substance release to the environment, an environmental transport medium for the substance, a point of receptor contact (exposure point) with the contaminated media, and an exposure route to the receptor at the exposure point."

A scoping report, presenting the results of the scoping assessment, is required. A standard checklist and scoping report outline are provided in the guidance document. At the end of a Level I scoping assessment, a decision is made to determine if no further ecological investigation is necessary or if the assessor should proceed to Level II.

Document: "Guidance for ecological risk assessment: Level II: B Screening" (Oregon DEQ 1997b).

The first step in a Level II screening assessment is to evaluate whether the information from the Level I scoping assessment is sufficient for a Level II problem formulation. If not, the assessor must conduct a site survey to supplement the Level I data. If there is sufficient information, the assessor proceeds to the next step, which is a site description update. This update is a more-detailed description and analysis of the ecological conditions at and near the site than that in Level I. After the site description is complete, site-specific ecological receptors must be identified, preferably for each habitat type. The COIs from the Level I assessment are then screened based on the frequency of detection, background concentration, toxicity criteria, and bioaccumulation potential of each compound to select contaminants of potential ecological concern (CPEC). The next steps are:

a. Identification of assessment end points.

b. Identification of complete exposure pathways.

c. Identification of known ecological effects.

From the above screening steps, a preliminary conceptual site model is developed and presented in both a graphical and narrative form. A report presenting the results of the Level II screening assessment must then be written, and a decision must be made as to the next course of action. At this level, there are three options:

1. No further action.

2. Response or remedial action.

3. Proceed to Level III.

"For a site to present a potential for risk, it must exhibit the following three criteria:

(a) contain CPECs in abiotic media at detectable and biologically significant concentrations,

(b) provide exposure pathways linking CPECs to ecological receptors, and

(c) have ecological receptors (those associated with assessment endpoints) that either utilize the site, are present nearby, or are in the locality of CPECs migrating from the site."

If there is no potential for risk, according to these criteria, then there is no further action or investigation is warranted.

The Oregon DEQ is currently working on a probabilistic risk assessment guidance document for human health risk assessment. The draft is currently being reviewed locally and should be available for wider review by the end of August 1997 (Oregon DEQ 1998). The State currently uses the EPA's CERCLA documents for human health risk assessments.

Texas

Document: "Texas Risk Reduction Program." Prepared by Texas Natural Resources Conservation Commission. (TNRCC 1996a).

Contact: TNRCC, Office of Waste Management, Austin, TX.

The Texas Risk Reduction Program for evaluation of human health risks follows the risk-based corrective action (RBCA) process developed by the American Society for Testing and Materials (ASTM). This document uses a tiered approach to determine risk-based concentration levels for certain contaminant levels at a hazardous waste site. These risk-based concentration levels are protective of human health. Also, the document specifies three "remedy standards" (risk management options) are given. The three options are:

1. Unrestricted Land Use - Permanent Remedy.

2. Restricted Land Use - Remedy with Controls.

3. No Active Land Use - Remedy with Controls.

The risk-based concentration levels are applied to one of these remedy standards to assess the risk to human health posed by a particular site. Three sections of this document (TNRCC 1996a) discuss procedures regarding determination of human health risk.

Section 4.4 describes how to determine human health risk limits and risk characterization.

The points of exposure (air, soil, groundwater, surface water) for each remedy standard are discussed in Section 5.4.

Section 7.1 explains the tiered process for the development of human health-based protective concentration levels.

In this tiered approach, the type of land use, remedy standards, and groundwater class specific to a site are first determined. The next step is a Tier I screening level evaluation. At each tier, exposure pathways and chemicals of concern, site parameters, protective concentration levels (PCLs) and PCL exceedance are determined. If a risk assessor cannot rule out the potential for harm to human health at the site in Tier I, he or she proceeds to a site-specific,

Tier II, risk assessment. Simple PLCs and analytical models are developed in a Tier II assessment. If PCLs are exceeded or risk cannot be ruled out at Tier II, the investigation proceeds to Tier III. Detailed, site-specific PCLs are derived in a Tier III assessment. Through this tiered approach, a cost-efficient remediation option that is protective of human health can be selected.

Document: "Guidance for conducting ecological risk assessments under the Texas Risk Reduction Program." (TNRCC 1996b).

Contact: TNRCC, Office of Waste Management, Austin, TX.

This guidance incorporates the tiered structure of the Texas Risk Reduction Program and is consistent with the USEPA's Framework for Ecological Risk Assessment. It also includes some modifications to make this document specific for Texas, such as the addition of state-developed criteria (Texas Surface Water Quality Standards) and the consideration of livestock and crops as potential ecological receptors.

As in the human health assessment process, the ecological risk assessment uses a three-tiered approach.

1. The purpose of Tier I is to characterize the site and identify potential exposure pathways.

2. Tier II is a screening-level ecological risk assessment, in which the contaminants at a site that are likely to pose an ecological risk are identified. There are three levels in a Tier II assessment:

    a. The first level compares established ecological benchmarks to the site data.

    b. The second level uses toxicity reference values derived from literature. This level involves problem formulation, an ecological effects evaluation, exposure estimates, and risk characterization.

    c. The third level reduces the Hazard Quotient by justifying the use of less conservative toxicity values than in the second level.

3. A Tier III assessment is a quantitative ecological risk assessment in which site-specific cleanup levels are developed.

A Tier III evaluation uses the effects and exposure information from the second level of a Tier II evaluation to develop a problem formulation. After the problem formulation, the study design is developed and verified in the field. Once a sampling plan is established, the samples can be collected and analyzed. The final step is a risk characterization, which can be used to select remediation alternative(s). Risk characterization involves risk estimation, risk description, and uncertainty analysis. The risk description of cleanup levels includes the threshold for effects on assessment end points as a range of values between the NOAEL (no observed adverse effects level) and the LOAEL (lowest observed adverse effects level) for a particular contaminant. Decisions regarding remediation, no further

action, or the need for further study are made by the risk manager at the completion of each tier.

Washington

The state of Washington does not have its own guidance document for conducting ecological risk assessments. Cleanup regulations for hazardous waste sites are currently being revised and a section on ecological risk will be incorporated. A similar revision of human health cleanup guidelines is also expected to occur. At this time, Washington does have documents regarding sediment cleanup standards. These documents include:

Documents: "Final environmental impact statement for the Washington State Sediment Management Standards" (December 1990). Chapter 173-204 WAC, Washington State Department of Ecology, Olympia, WA.

"Summary of guidelines for contaminated freshwater sediments" (March 1995). Publication No. 95-308, Washington State Department of Ecology, Olympia, WA.

"Review and evaluation of Microtox® test for freshwater sediments" (November 1992). J. Bennett and J. Cubbage, ed., Washington State Department of Ecology, Olympia, WA.

"Sediment management standards" (December 1995).

Contact: Nigel Blakely, Department of Ecology, Toxic Cleanup Program, Technical Policy Department.

Appendix B
Food Chain and Toxicity Models

This appendix provides brief descriptions of several food chain and toxicity models for use in ecological risk assessment. These include:

a. Gobas Food Chain Model

b. Great Lakes Methodology for Predicting Fish Tissue Concentrations from Water Concentrations

c. Sum polycyclic aromatic hydrocarbon (PAH) Model

d. Narcosis Model

Gobas Food Chain Model

The Gobas model estimates the bioaccumulation and biomagnification of organic contaminants (except PAHs) in aquatic food web due to surface water, sediment, and food web exposure. The model is most useful for predicting the concentrations of organic compounds that are not readily metabolized. It uses compound-specific information, including the octanol-water partition coefficient, K_{ow}, and Henry's Law Constant to predict the disposition of contaminants in an aquatic food web.

A few compounds, for which Henry's Law Constants are not available, cannot be modeled with the Gobas model (PCB-183, -184, and -185, o,p-DDD, o,p-DDE, and trans-nonachlor).

The Gobas model consists of five major compartments: phytoplankton, zooplankton, benthic invertebrates, forage fish, and piscivorous fish. Concentrations of chemicals of concern (COCs) in the phytoplankton, zooplankton, and benthic invertebrates (polychaetes and mysid shrimp) are estimated using equilibrium partitioning from water and sediment to biota (BSAF=1). Concentrations in forage fish and piscivorous fish are estimated using mass-transfer coefficients that describe uptake of chemical from water and ingestion of organisms from lower trophic levels, elimination of contaminant by excretion, and dilution by growth.

Mass transfer coefficients are estimated on the basis of empirical relationships between organism wet weight, lipid content, and percentage of each prey item in the diet. The model conservatively assumes that there is no loss of compound due to metabolic transformation.

Information on the food chain of the management site is used to describe and select typical organisms for use in the model.

Great Lakes Methodology for Predicting Fish Tissue Concentrations from Water Concentrations.

The U.S. Environmental Protection Agency (EPA) has relied upon use of steady-state bioconcentration factors (BCF) and bioaccumulation factors (BAF) to relate water concentrations to the concentrations in fish tissue. A steady-state BCF describes the ratio (L/kg) of a compound's concentration in tissue to its concentration in the surrounding water, when the organism is exposed in the laboratory only through the water, by uptake through gill membranes or other external body surfaces.

$$BCF = \frac{(\mu g\ COC/kg\ wet\ wt\ tissue)}{(\mu g\ COC\ /\ L\ water)} = L/kg$$

A steady state BAF describes the ratio (L/kg) the concentration of a substance in tissue to its concentration in the surrounding water in situations where both the organism and its food are exposed. BAF are typically based on field measurements.

$$BAF = \frac{(\mu g\ COC/kg\ wet\ wt\ tissue)}{(\mu g\ COC\ /\ L\ water)} = L/kg$$

If lab or field measurements are unavailable, USEPA recommends that the following methodology be used to derive BCFs and BAFs.

BCFs for organic compounds can be calculated from the octanol-water partition coefficient, K_{ow}, using the following relationship (Veith and Kosian 1983):

$$\log BCF = 0.79 \log K_{ow} - 0.40$$

In the absence of a field measured BAF, USEPA recommends estimating BAFs for organic compounds by multiplying the BCF by a factor which accounts for the biomagnification of a pollutant through the food chain and lipid content of the organism. As larger organisms, such as bluefish, consume other fish and aquatic organisms, the concentration of some COCs are increased in the predator. The factor which describes this biomagnification is called the food chain multiplier (FCM). USEPA calculated FCMs that describe biomagnification

through the top predatory fish in the food chain, trophic level 4 (Thomann 1989). Estimation of FCMs for organic contaminants with K_{ow} greater than 6.5 is less certain and for such compounds, the USEPA recommends using a FCM of 1 as a default value.

For lipophilic organic chemicals, BCFs and BAFs are presumed to be directly proportional to the percent lipid from one tissue to another, and BAF are calculated as follows:

$$\text{Wildlife BAF} = (\text{predicted BCF})(Ol/Fl)(FCM)$$

$$\text{Human Health BAF} = (\text{predicted BCF})(FFl/F1)(FCM)$$

where

predicted BCF (L/kg) is estimated from the regression described above (not to exceed 100,000)

Fl = average percent lipid of the organisms used to establish the relationship between BCF and K_{ow}

Ol = percent lipid content of the receptor

FFl = average percent lipid content for a fish fillet

FCM = appropriate food chain multiplier

Concentrations of metals in fish tissue can be estimated from established BAFs by using a methodology recommended by the EPA (Stephan 1993). Established BAFs for metals are based on measured BCFs and BAF and are not calculated with FCM.

Fish tissue concentrations of both organic contaminants and metals are calculated as:

$$C_F = (C_W)(BAF)$$

where

C_F = concentration of contaminant in fish (g COC/kg wet wt)

C_w = concentration of contaminant in water (g COC/L)

BAF = appropriate bioaccumulation factor (L/kg)

Sum-PAH Model

Polycyclic aromatic hydrocarbons (PAHs) can be biotransformed by aquatic organisms to metabolites that exert toxic effects by more specific modes of action than nonpolar narcosis. A concentration-response model has been developed which predicts toxic effects of PAHs to benthic invertebrates (Swartz et al. 1995).

The model uses a regression, based on the relationship between concentrations of PAHs in interstitial water and toxicity to estuarine amphipods in spiked sediment bioassays, to determine toxic units for PAHs. Toxic units for each compound, which are equal to the concentration in the interstitial water of the contaminated sediment divided by the interstitial water 10-d LC50, are summed. The sum of the toxic units is used to predict the probability of significant acute sediment toxicity to marine and estuarine amphipods, where significant mortality was defined as >24 percent mean mortality in field-collected sediments. The model was verified by comparison to mortality observed in sediment toxicity tests in field-collected samples from 13 investigations. The Sum-PAH (Σ-PAH) level of acute toxicity (Sum of Toxic Units = 3.291, p of >24% mortality = 1.0) was determined as the toxic-unit concentration above which acute toxicity is always expected to occur. The Σ-PAH threshold of acute toxicity (Sum of Toxic units = 0.186, p of >24% mortality = 0.05) is the toxic unit concentration below which mixtures of PAHs are unlikely to contribute to sediment toxicity above background. The 50 percent probability of acute toxicity (sum of toxic units = 0.725, p>0.050) is the concentration expected to cause acute toxicity in 50 percent of the cases.

Narcosis

Narcosis due to organic contaminants in aquatic organisms is defined as a nonspecific reversible disturbance in the functioning of the membrane, caused by the accumulation of contaminants in the hydrophobic (lipid) phases of the organism (van Wezel and Opperhuizen 1995). Experimental work has demonstrated that the critical body residue (CBR) for the acute lethal effect of nonpolar narcotic chemicals is fairly constant at 2 to 8 mmol chemical/kg wet wt tissue (McCarty et al. 1992). In addition, the effects of mixtures of nonpolar chemicals that act by a narcotic mode of action appear to be generally additive (McCarty and Mackay 1993).

Appendix C
Information Sources

These sections describe those sources of information which may be incorporated into risk assessments. They include:

a. Periodically published information bulletins on risk assessment from Government agencies.

b. Updated agency databases such as Integrated Risk Information System (IRIS) database, Health Effects Assessment Summary Tables (HEAST) database, and Acquire.

c. WEB sites regarding chemical, biological, physical, and engineering information useful to development of risk assessment - descriptions of web sites such as U.S. Geological Survey (USGS), U.S. Environmental Protection Agency (USEPA), National Oceanic and Atmospheric Administration (NOAA), U.S. Army Corps of Engineers (USACE) which provide specific data and literature reviews of information useful to conducting risk assessments.

Periodically Published Information Sources

This section identifies periodically published information sources concerning human health and ecological risk assessment from Federal and state government agencies. It provides a brief description of the information source, the publishing agency, and the availability of each bulletin.

Agency Databases

This section identifies and describes those Federal and state databases used in developing human-health and ecological risk assessments. It includes information on how to access the databases and where in a risk assessment to incorporate the information in a specific database.

WEB Sites

This section provides descriptions and electronic addresses for WEB sites which contain information useful in conducting risk assessments. These include sites maintained by Federal agencies, state environmental agencies, and professional societies. The section is not exhaustive, but the reader should be aware that most of these sites maintain links to other relevant sites. For each identified site, this subsection provides a brief description of the categories of information available.

Federal Agencies

Agency: United States Army Corps of Engineers/Waterways Experiment Station Environmental Lab
Internet Address: *www.wes.army.mil/el/homepage/html*
Description:
This site describes the Environmental Laboratory, the research staff, and its mission. What's New, WES Maps, and a description of corps training can be accessed from this Web Site.

Agency: United States Environmental Protection Agency
Internet Address: *www.epa.gov/epahome*
Description:
Particular sites of interest that can be accessed from the USEPA's homepage are listed. Also, you can reach the homepage of each USEPA region by selecting "Offices and Regions" at the USEPA homepage. A map of the United States will appear and you can click on the region of interest and view the homepage for that region.

Agency: IRIS (Integrated Risk Information System)
Internet Address: *www.epa.gov/ngispgm3/iris*
Description:
"IRIS is an electronic database containing information on human health effects that may result from exposure to various chemicals in the environment." IRIS contains chemical files containing information such as oral reference doses and inhalation reference concentrations for chronic noncarcinogenic health effects and hazard identification, oral slope factors, and oral and inhalation unit risks for carcinogenic effects. The database also contains supporting information such as a description of the rationales and methods used to develop the values described above, a discussion of the limitations to the use of information in IRIS, and a glossary of terms and acronyms used in the chemical files. The chemicals are listed alphabetically and are searchable by name or by Chemical Abstracts Service Registry Number (CASRN).

Agency: National Center for Environmental Assessment (NCEA)
Internet Address: *www.epa.gov/ncea*
Description:
This site provides information about ecological risk assessment guidance and methods and chemical-specific human health risk assessments conducted by the USEPA. Also at this site are links to other EPA and non-EPA websites. The NCEA is part of the Office of Research and Development.

Agency: 1995 National Listing of Fish Consumption Advisories
Internet Address: *www. epa.gov/OST/fishadvice*
Description:
"This database includes all available information describing State-issued fish and wildlife consumption advisories for the 50 States, the District of Columbia, and four U.S. Territories."

Agency: Office of Research and Development
Internet Address: *www.epa.gov/ORD*
Description:
Of note at this site are the Offices and Laboratories of the ORD and special EPA and non-EPA website links.

Agency: Office of Science and Technology, Office of Water
Internet Address: *www.epa.gov/OST*
Description:
OST publications of water quality standards, sediment quality criteria, drinking water criteria, criteria for contaminated sediments, etc... are available at this site.

Agency: USEPA R/V Mudpuppy: Background on Contaminated Sediments in the Great Lakes (from the Great Lakes National Program Office)
Internet Address: *www.epa.gov/glnpo/sediment/mudpup.html*
Description:
This site provides information about the Great Lakes National Program Office and the services of the Mudpuppy, its 32-foot flat-bottom boat designed for sediment sampling in shallow rivers and harbors. It describes two Great Lakes projects using the Mudpuppy. Assessment and remediation of contaminated sediments (ARCS) reports are available at this site.

State agencies

Agency: Massachusetts Department of Environmental Protection (DEP)
Internet Address: *www.magnet.state.ma.us/dep/dephome.htm*
Description:
The Massachusetts DEP website provides information about the organization of the DEP (contacts, offices) and how to obtain copies of DEP regulations. This site also has links to other related environmental sites on the World Wide Web.

Agency: Office of Research and Standards (ORS)
Internet Address: *www.magnet.state.ma.us/dep/ors/orshome.htm*
Description:
"The Office of Research and Standards was created in 1980 to provide DEP with information on the adverse impacts of environmental contaminants and to make recommendations for protecting public health and the environment. ORS personnel are highly-trained scientists in areas of toxicology, risk assessment, chemistry, public health, ecology and biology."

Professional Organizations

Organization: Air and Waste Management Association (AWMA)
Internet Address: *www.awma.org*
Description:
The Air and Waste Management Association provides a forum for exchange of viewpoints on technical, scientific, economic, social, political, and risk assessment environmental issues. The organization has more than 16,000 members in 65 countries representing many disciplines: physical and social sciences, health and medicine, engineering and law. The AWMA produces a variety of publications including a peer-reviewed journal, a news magazine, periodicals, books, preprints of technical papers, training manuals, and a monthly membership newsletter. These publications provide important information about environmental decision-making worldwide.

Organization: American Industrial Hygiene Association (AIHA)
Internet Address: *www.aiha.org*
Description:
This site features information on Occupational and Environmental Health and Safety issues. You may access information at this site regarding AIHA scientific affairs, professional development, products and publications, and other resources.

Organization: American Society for Testing and Materials (ASTM)
Internet Address: *www.astm.org/PUB*
Description:
"ASTM has developed and published 10,000 technical standards, which are used by industries worldwide. ASTM members develop the standards within the ASTM consensus process." This Web Site allows you to search for standards of interest (e.g., human health toxicological profiles, indoor air default values, water quality standards).

Organization: American Society of Limnology and Oceanography
Internet Address: *aslo.org*
Description:
This site provides access to DIALOG (Dissertations Initiative for the Advancement of Limnology and Oceanography)

Organization: American Water Resources Association
Internet Address: *www.awra.org*
Description:
This site links you to the Universities Water Information Network.

Organization: Environmental Research and Technology in Gas Research Institutes GRI/Net
Internet Address: *www.gri.org/signpost.htm*
Description:
"The Gas Research Institute's (GRI) Environmental Technology and Information Center (ETIC) communicates information about GRI-sponsored environmental research and technology." By entering a key word or phrase in the search box at this site, you can search for information on the management of manufactured gas plant sites, air toxics emissions from gas-fired combustion sources, glycol dehydrator emissions, mercury contamination at gas-metering sites, and other environmental content of potential interest to the gas industry.

Organization: Environmental Sciences Division at Oak Ridge National Laboratory
Internet Address: *www.esd.ornl.gov.*
Description:
This site describes the Oak Ridge National Laboratory (ORNL) Environmental Sciences Division (ESD) and its major programs, its facilities, publications, and projects.

Organization: Estuarine Research Federation (ERF)
Internet Address: *www.erf.org*
Description:
The Estuarine Research Federation Homepage describes its resources and programs. This organization promotes research in estuarine and coastal waters, and promotes communication between members of affiliated societies.

Organization: ESTUARIES (Journal of the ERF)
Internet Address: *www.erf.org/journal/journal.html*
Description:
This Web Site describes the Journal of the Estuarine Research Federation. Estuaries is abstracted or indexed in BIOSIS; Oceanic Index; Current Titles in Ocean, Coastal, Lake & Waterway Sciences; Meteorological & Geophysical Abstracts, and others.

Organization: Harvard School of Public Health Center for Risk Analysis
Internet Address: *www.hsph.harvard.edu/organizations/hcra/hcra.html*
Description:
The Harvard School of Public Health Center for Risk Analysis Web Page describes the center, its degree programs and courses, and provides many links to other departments in the school.

Organization: Massachusetts Licensed Site Professional Association (LSPA)
Internet Address: *www.lspa.org/reference/links.htm*
Description:
"The LSPA operates this World Wide Web site to assist LSPs and environmental consultants in staying current with the LSP Regulations, the Massachusetts Contingency Plan (MCP), and environmental matters in general." This site contains several links to risk assessment organizations and web sites used to gather information for risk assessments.

Organization: National Shellfisheries Association (NSA)
Internet Address: *www.shellfish.org*
Description:
The National Shellfisheries Association is an "international organization of scientists, management officials and members of industry, concerned with the biology, ecology, production, economics and management of shellfish resources B clams, oysters, mussels, scallops, snails, shrimp, lobsters, crabs, among many other species of commercial importance." The NSA publishes the Journal of Shellfish Research as well as a Quarterly Newsletter which are available to view on-line.

Organization: National Status and Trends Program, Mussel and Mollusk Watch
Internet Address: *www-orca.nos.noaa.gov/projects/nsandt/nsandt.html*
Description:
The National Oceanic and Atmospheric Administration's National Ocean Service, Office of Ocean Resources Conservation and Assessment (ORCA), has a National Status and Trends Program that, since 1984, has monitored spatial and temporal distributions of chemical contamination and biological responses to that contamination" across the United States. "Temporal trends are being monitored through the Mussel Watch project that analyzes mussels and oysters collected annually at about 200 of those sites. Spatial trends have been described on a national scale from chemical concentrations measured in surface sediments collected by both the Mussel Watch and The Benthic Surveillance Projects from 240 sites distributed throughout the coastal and estuarine United States." The raw data from these projects are available on-line.

Organization: RiskWORLD
Internet Address: *www.riskworld.com*
Description:
RiskWORLD has links to many risk assessment and risk management sites.

Organization: Society for Risk Analysis
Internet Address: *www.sra.org*
Description:
"The Society for Risk Analysis (SRA) provides an open forum for all those interested in risk analysis. Risk analysis is broadly defined to include risk assessment, risk characterization, risk communication, risk management, and policy relating to risk...Our membership is multidisciplinary and international." The website of the SRA features links to sites of scientific societies or

nongovernmental organizations with interests related to risk analysis. The SRA Web also has a "risk science" section which provides information on the substance of risk analysis.

Organization: Society for Sedimentary Geology (SEPM)
Internet Address: *www.ngdc.noaa.gov/mgg/sepm/sepm.html*
Description:
"SEPM is an international not-for-profit Society based in Tulsa, Oklahoma, dedicated to the dissemination of scientific information on sedimentology, stratigraphy, paleontology, environmental sciences, marine geology, hydrogeology, and many additional related specialties." Articles in the two journals published by SEPM, the Journal of Sedimentary Research and PALAIOS, are available on-line at this site.

Organization: Society of Environmental Toxicology and Chemistry (SETAC)
Internet Address: *www.setac.org*
Description:
Featured at this site are SETAC publications related to ecological risk assessments. Examples of such publications include Ecological Risk Assessments of Contaminated Sediments and Ecotoxicological Risk Assessment of the Chlorinated Organic Chemicals.

Organization: Society of Wetlands Scientists (SWS)
Internet Address: *www.sws.org*
Description:
Recent volumes of the Journal of SWS are available on-line at this site. Also, links to other wetlands sites can be made using the following uniform resource locator (URL): *www.sws.org/wetlandweblinks.html*

Organization: United States Geological Survey- National Geospatial Data Clearinghouse
Internet Address: *nsdi.usgs.gov/nsdi/products/water_data.html*
Description:
This site provides water quantity and quality data for geographic regions of the United States. Examples of water data include "stream discharge (flow), stage (height), reservoir and lake stage and storage, groundwater levels, well and spring discharge, and the quality of surface and groundwater."

Organization: Universities Water Information Network (UWIN)
Internet Address: *www.uwin.siu.edu*
Description:
"The Universities Water Information Network disseminates information of interest to the water resources community and all concerned with our water resources. UWIN is housed at the Headquarters of the Universities Council on Water Resources at Southern Illinois University in Carbondale, Illinois. UWIN is funded through a grant from the United States Geological Survey and is part of their outreach efforts to the water resources community."

Appendix D
Toxicological Profiles

Acenaphthene
Cas No. 83-32-9

Potential sources and exposure

Acenaphthene is a polycyclic aromatic hydrocarbon (PAH). The reader should refer to the general profile on PAHs for exposure information.

Physical and chemical properties

Property	Value
Molecular weight	154.2 g/mol
Water solubility	3.42 mg/L at 25 °C
Vapor pressure	1.55×10^{-3} mm Hg at 20 °C
K_{oc}	4,600 mL/g
log K_{ow}	4
Henry's Law Constant	9.1×10^{-5} atm-m^3/mole

Toxicity

Acenaphthene has been shown to be irritating to the skin and mucous membranes and to cause vomiting following ingestion.

A review of the reported literature indicates that there are no conclusive experiments demonstrating the carcinogenic potential of acenaphthene. Studies using several different bacterial test systems provide no evidence of mutagenicity. No information concerning its teratogenicity or reproductive toxicity is available.

The oral RfD of 0.06 mg/kg-day for acenaphthene is based on subchronic study in mice. Four groups of CD-1 mice (20/sex/group) were gavaged daily with acenaphthene for 90 days. Liver weight changes accompanied by

microscopic alterations (cellular hypertrophy) were noted in both mid- and high-dose animals and seemed to be dose-dependent. The lowest observed adverse effects level (LOAEL) of 350 mg/kg/day is based on hepatotoxicity; the no observed adverse effects level (NOAEL) is 175 mg/kg/day.

Toxicokinetics

Like other PAH compounds, acenaphthene is oxidized by liver enzymes to form water-soluble derivatives that can be excreted in urine. No data were located on the absorption of acenaphthene in laboratory animals or humans. In the absence of data, it is assumed that 100 percent of acenaphthene is absorbed via the oral or inhalation exposure routes.

Ecological effects

In aquatic acute toxicity tests EC50 values of 41,200 and 1,700 ug/L are reported for the cladoceran Daphnia magna and the bluegill, respectively. In saltwater species, the acute toxicity (96-hr LC50) values for shrimp and sheepshead minnow are 970 ug/L and 2,230 ug/L, respectively. A chronic value of 710 ug/L is reported for the sheepshead minnow, yielding an acute:chronic ratio of 3:1.

A bioconcentration factor of 387 has been determined for bluegill sunfish.

A study summarizing the toxicity of a variety of compounds to wild and domestic bird species indicates that the LD50 of acenaphthene for redwinged blackbird is greater than 100 mg/kg.

Acenaphthylene
Cas No. 208-96-8

Potential sources and exposure

Acenaphthylene is a PAH. The reader is referred to the general profile on PAHs for exposure information.

Physical and chemical properties

Property	Value
Molecular weight	152.2 g/mol
Water solubility	3.93 ppm at 24 °C
Vapor pressure	2.9×10^{-2} at 20 °C
K_{oc}	2,500 mL/g
log K_{ow}	3.7
Henry's Law Constant	1.48×10^{-3} atm-m^3/mol

Toxicity

Little information regarding the acute or chronic toxicity of acenaphthylene is available.

There are no long-term studies in the literature that adequately evaluate the carcinogenicity of acenaphthylene, nor are there any data from epidemiologic studies which correlate acenaphthylene exposure with an increased risk of cancer. A skin-painting study in mice produced negative results (IRIS 1992). Structurally, acenaphthylene is similar to other low molecular weight polycyclic aromatic hydrocarbons that are considered to be noncarcinogenic. Acenaphthylene is classified as a Group D carcinogen by the U.S. Environmental Protection Agency (USEPA) based on the lack of human carcinogenicity data and inadequate data from animal bioassays.

Positive results have been reported from a single mutagenicity test in which acenaphthylene was tested in a strain of Salmonella typhimurium in the presence of liver microsomal activation (USEPA 1982). Other tests in Salmonella have been negative (IRIS 1992). There is currently no RfD for acenaphthylene, although based on structure-activity relationships with anthracene, an oral RfD of 0.3 mg/kg-day is recommended.

Toxicokinetics

Like other PAH compounds, acenaphthylene is oxidized by liver enzymes to form water-soluble derivatives that can be excreted in urine. No data were

located on the absorption of acenaphthylene in laboratory animals or humans. Because of their high lipid solubility, PAHs are believed to be distributed throughout the body. Relative to other tissues, they tend to localize in body fat and fatty tissues.

Ecological effects

The reader is requested to review the toxicity profile for the PAHs for information regarding ecological effects. A no effect level of 5 mg/L was observed for trout in an acute (24 hr) exposure. Adequate data for characterization of toxicity to domestic animals and wildlife are not available.

References

Integrated Risk Information System (IRIS) on-line database. (1992).

U.S. Environmental Protection Agency. (1982). "An exposure and risk assessment for benzo(a)pyrene and other polycyclic aromatic hydrocarbons," Volume IV, Final draft report, Washington, DC.

Anthracene
(Paranaphthalene)
Cas No. 120-12-7

Potential sources and exposure

Anthracene is a PAH. The reader should refer to the general profile on PAHs for exposure information.

Physical and chemical properties

Property (a)	Value	Reference
Molecular weight	178.2	Mabey et al. (1982)
Water solubility	1.29 ppm at 25 °C 4.5×10^{-2} ppm at 25 °C	Verschueren (1983) Mabey et al. (1982)
Vapor pressure	1.7×10^{-5} mmHg at 20 C	Mabey et al. (1982)
K_{oc}	1.4×10^{4}	Mabey et al. (1982)
K_{ow}	2.8×10^{4}	Mabey et al. (1982)
Henry's Law Constant	8.6×10^{-5} atm^{-3}/mol at 25 °C	Mabey et al. (1982)

Different values for the physical and chemical properties of various compounds are reported in the literature by different sources. The values differ typically because the experiments used to determine them were performed under different conditions (e.g., temperature). For more information about the properties of various compounds, the investigator should consult the different databases that have been compiled such as the Integrated Risk Information System (IRIS) that is available from the USEPA.

Toxicity

No epidemiological studies were identified which examined possible human health effects resulting from exposure to anthracene. Few reports of health effects in humans resulting from anthracene exposure exist. It is reported that three cases of epithelioma (any tumor derived from epithelium) of the hand, cheek, and wrist occurred in men handling crude anthracene in an alizarin factory (Kennaway 1924 as cited in International Agency for Research Center 1983). In another instance it was reported that in studies on the treatment of psoriasis, anthracene solubilized in an alcohol N-methyl-2-pyrrolidone vehicle, induced photosensitive reactions when administered topically in low concentrations (~O.25%) to humans in combination with ultraviolet (UV) radiation (Urbanek 1980 and Walter 1980 as cited in IARC 1983).

Anthracene has been tested for carcinogenicity in a number of different species, using a variety of routes of administration, with primarily negative results. There is no evidence that anthracene is active in short-term tests. IARC (1983) concludes that the available data provide no evidence that anthracene is carcinogenic to experimental animals.

Toxicokinetics

In the review of polycyclic aromatic hydrocarbons, the USEPA (1982) notes that anthracene appears to be converted to 1,2-dihydroanthracene-1,2-diols and their glucuronides. In an investigation in which anthracene was incubated with rat liver preparations (Akhtar et al. 1979 as cited in IARC 1983), the major metabolite was identified as the 1,2-dihydrodiol. It has also been reported that the 1,2-dihydrodiol, 9,10-anthraquinone, 9,10-dihydrodiol, and 2,9,10-trihydroxyanthracene have been identified as metabolites in rat urine, together with conjugates consistent with the formation of the 1,2-oxide (Sims 1964 as cited in IARC 1983).

Ecological effects

The profile for benzo(a)pyrene provides a generic description of the potential environmental effects of PAHs as a class of compounds. A no-effect level of 5 mg/L was observed for trout in an acute (24-hr) exposure. Adequate data for characterization of toxicity to domestic animals and wildlife are not available.

Reported levels in sediments:

	mg/kg
Penobscot Bay, ME, outer region	0.0069
Buzzards Bay, New Bedford, MA	0.0070 - 0.0080
Penobscot Bay, ME, inner region	0.0234
New York Bight	0.0391
The Graves, Boston MA	0.0420
Boston Harbor	0.0725
Buzzards Bay New Bedford, MA	0.1700
Boston Harbor Aquarium/Fort Point	0.2450
Boston Harbor	0.2833
Buzzards Bay, New Bedford, MA	0.3400
Chelsea River, MA	0.4110
Long Island Sound	0.4550
Savern Estuary, U.K.	02.4

Reported levels in soils:

	Concentration (mg/kg)
Anthracene	0.008-0.017

Reported levels in air:

Averages for	
Residential	0.03-0.83
Rural	0.4
Urban	0.068-0.278
Urban	0.1-1.3
Detroit	1.2

References

International Agency for Research on Cancer (IARC). (1983). "IARC monographs on the evaluation of the carcinogenic risk of chemicals to humans." *Polynuclear aromatic compounds, Part 1, chemical, environmental and experimental data.* Vol 32, World Health Organization, Lyon, France.

Mabey, W. R., Smith, J. H., Podell, R. T., Johnson, H. L., Mill, T., Chou, T. W., Gates, J., Partridge, I. W., Jaber, H., and Vandenberg, D. (1982). "Aquatic fate process data for organic priority pollutants," Prepared by SRI International for U.S. Environmental Protection Agency, Office of Water Regulations and Standards, Monitoring and Data Support Division, USEPA Contracts 68-01-3867 and 68-03-2981, Washington, DC.

U.S. Environmental Protection Agency. (1982). "An exposure and risk assessment for benzo(a)pyrene and other polycyclic aromatic hydrocarbons: Vol III, anthracene, acenaphthene, fluoranthene, fluorene, phenantherene, and pyrene," Final Draft Report, Office of Water Regulations and Standards, Washington, DC.

Verschueren, K. (1983). *Handbook of environmental data on organic chemicals.* 2nd ed., Van Nostrand Reinhold Co., New York.

Arsenic
Cas No. 7440-38-2

Potential sources and exposure

Arsenic is a naturally occurring metal that has been widely used in rat and ant poisons, herbicides, some medicines, and in arsenic treated (pressure treated) wood. Some areas of the United States have unusually high natural levels of arsenic in rock, which can lead to high concentrations in soil and water. Most foods contain a low level of arsenic; however, seafood and freshwater fish contain elevated levels of arsenic. There are several forms of arsenic to which an individual might be exposed and the toxicity is dependent upon the type of arsenic compound.

Physical and chemical properties

Property	Value
Molecular weight	74.92 g/mol
Water solubility	insoluble at 25 °C

Toxicity

The toxicity of arsenic depends upon its chemical form and route, dose, and duration of exposure. In general, arsenites (As^{3+}) are more toxic than arsenates, soluble arsenic compounds are more toxic than insoluble compounds, and inorganic arsenic compounds are more toxic than organic derivatives (Agency for Toxic Substances and Disease Registry (ATSDR) 1992).

Arsenic is an irritant of the skin, mucous membranes, and gastrointestinal tract. Symptoms of acute toxicity include vomiting, diarrhea, convulsions, and a severe drop in blood pressure. Subchronic exposures may result in hyperpigmentation of the skin, persistent headache, and lethargy. Chronic exposures to inorganic arsenic compounds may lead to neurotoxicity of both the peripheral and central nervous systems as well as peripheral vascular disease and skin lesions.

The most potent forms of the compound are the trivalent arsenic compounds. These compounds can bind to sulfhydral groups on proteins and enzymes. Arsenic affects mitochondrial enzymes and impairs tissue respiration, which seems to be related to the cellular toxicity (Klaassen, Amdur, and Doull 1995). Arsenic compounds are inducers of metallothionein which can serve a detoxicating function.

The USEPA classifies arsenic as a known human carcinogen based on epidemiological studies in which a causal association between exposure and skin

cancer was observed in Taiwanese and Chilean populations exposed to arsenic in drinking water (IRIS 1999).

Toxicokinetics

Arsenic (trivalent or pentavalent insoluble forms) is well absorbed from the gastrointestinal tract. Limited data suggest nearly complete absorption of soluble forms of trivalent and pentavalent arsenic. Deposition of arsenic in the airway is dependent on particle size and chemical form. Excretion of absorbed arsenic is mainly via the urine. Arsenic has a predilection for the skin and is excreted by desquamation of skin and in sweat, particularly during periods of profuse sweating. It also concentrates in nails and hair. Dimethyl arsenic is the principal detoxication product (Klaassen, Amdur, and Doull 1995).

Ecological effects

Bioaccumulation. Arsenic is neither a major contaminant of aquatic plants nor does it normally concentrate in either freshwater or marine fish. Only in extreme cases of ambient pollution does it contaminate aquatic plants and there are few reports of tissue residues exceeding health guidelines in fish. However, some reports do demonstrate rather high levels in invertebrates, for example, exceeding 30 mg/kg.

Toxic effects to aquatic organisms. Although insufficient data exist to determine the definitive acute toxicity to organisms, fresh or marine, work on the topic indicates that large doses of arsenic (greater than 1 mg As/L) are required to induce acute toxic effects in both plants and invertebrates. Chronic effects for both invertebrates and fish exposed to inorganic arsenic have been reported and require a relatively large dose, typically > 5 mg As/L.

Toxic effects to wildlife (tertiary). To be absorbed by terrestrial plants, arsenic compounds must be in a mobile form in the soil. Unless located in an area where arsenic concentrations are exceptionally high, plants will distribute accumulated arsenic in nontoxic amounts throughout the plant body. Most plants will yield significantly less of a crop when concentrations become 3 to 28 mg/L of water soluble arsenic and 25 to 85 mg/kg of total arsenic. Air concentrations up to 3.9 Fg As/m^3 have also been seen to have adverse effects on vegetation.

Effects on soil biota and insects remain limited, but generally it is believed that soil microorganisms are capable of tolerating relatively high concentrations of arsenic.

In birds, signs of inorganic trivalent arsenite poisoning include muscular uncoordination, debility, slowness, jerkiness, falling hyperactivity, fluffed feathers, drooped eyelid, huddled position, immobility, and seizures. Studies suggest that lethal acute inorganic arsenic poisoning results in the destruction of blood vessels lining the gut, thereby causing decreased blood pressure and subsequent shock.

Mammalian exposure to arsenic occurs primarily through ingestion. Acute episodes of poisoning are characterized by high mortality and morbidity. Signs of arsenic toxicosis include intense abdominal pain, staggering gait, extreme weakness, trembling, salivation, vomiting, diarrhea, prostration, collapse, and death. Chronic poisoning is infrequently seen due to the fact that excretion and detoxification are rapid.

References

Agency for Toxic Substances and Disease Registry (ATSDR). (1992). "Toxicological profile for arsenic," U.S. Public Health Service, Washington, DC.

Integrated Risk Information System (IRIS). (1999). On-line database, accessed 1999.

Klaassen, Curtis D., Amdur, Mary, and Doull, John. (1995). *Toxicology: The basic science of poisons.* 5th ed., McGraw-Hill, New York.

Benzo(a)anthracene (Benz(a)anthracene; 1,2-Benz(a)anthracene; Benzo(a)phenanthrene) Cas No. 56-55-3

Potential sources and exposure

Benzo(a)anthracene is a PAH. The reader should refer to the general profile on PAHs for exposure information.

Physical and chemical properties

Property	Value
Molecular weight	228.3 g/mol
Water solubility	5.7×10^{-3} mg/L at 20 °C
Vapor pressure	2.20×10^{-8} mm Hg at 20 °C
K_{oc}	1,380,000 mg/L
log K_{ow}	5.6
Henry's Law Constant	1.16×10^{-6} atm-m^3/mol

Toxicity

Although there are no human data that specifically link exposure to benzo(a)anthracene to human cancers, benzo(a)anthracene is a component of mixtures associated with human cancer. These include coal tar residues, coke oven emissions, and cigarette smoke (IRIS 1992).

Several studies indicate that benzo(a)anthracene is carcinogenic in animals, and IARC has evaluated that evidence as sufficient to establish the carcinogenicity of benzo(a)anthracene in animals (IARC 1983). Benzo(a)anthracene administration caused an increase in the incidence of tumors by gavage, dermal application (IARC 1973); and both subcutaneous injection, and intraperitoneal injection assays. A carcinogenic potency factor (CPF) has not been developed by the USEPA. Based on the work of Bingham and Falk (1969), it was estimated that benzo(a)anthracene has a relative potency - to benzo(a)pyrene - of 0.145. (Potency is approximately 14.5 percent of that of benzo(a)pyrene). This value can be used in the relative potency approach for estimating carcinogenic risk.

Extensive testing for mutagenicity has been documented (IARC 1983) with mostly positive results (IRIS 1992).

Toxicokinetics

Some benzo(a)anthracene metabolites have been shown to induce mutations, cell transformation, and to bind to nucleic acids. The metabolites of benzo(a)anthracene are mutagenic and tumorigenic (IARC 1983).

Nucleic acid (DNA) adducts are formed in the skin from the metabolites 3,4-diol-1,2-epoxide and 8,9-diol-10,11-epoxide (IARC 1983). No information is available regarding dermal or oral absorption coefficient, although benzo(a)anthracene was reported to be readily transported across the gastrointestinal mucosa (USEPA 1984).

Benzo(a)anthracene induced benzo(a)pyrene hydroxylase in rat placenta (IARC 1983).

Ecological effects

The reader is requested to review the toxicity profile for PAHs for information regarding ecological effects.

Hinga et al. (1980) examined the biogeochemistry of C-14 labeled benzo(a)anthracene in an enclosed marine ecosystem. The experiment was conducted for 230 days. At the end of the experiment, 29 percent of the chemical had been respired to CO_2, while the remaining extractable activity (43 percent) was evenly divided between parent compound and intermediate metabolic products. Total C-14 activity was removed from the water with a half-life of about 52 hr, while the C-14 parent compound had a half-life of 24 hr. The chemical became associated with the sediments and was mixed deeper into the sediments by benthic animal activity. The authors made a rough calculation of the half-life in sediments and noted stated that half-lives on the order of 1.2 to 3 years may be calculated. They further point out, however, that the occurrence of benzo(a)anthracene at some depth in natural sediments suggests that a fraction of the compound and perhaps some of its metabolites may persist indefinitely.

References

Bingham, E., and Falk, H. L. (1969). "Environment carcinogens, The modifying effect of carcinogens on the threshold response," *Arch. Environ. Health* 19, 779-783.

Hinga, K. R., Pilson, M., Lee, R. F., Farrington, J. W., Tjessem, K. and Davis, A. C. (1980). "Biogeochemistry of benzoanthracene in an enclosed marine ecosystem," *Envir. Sci. & Tech.* 14, 1136-1143.

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International Agency for Research on Cancer (IARC). (1983). "Monographs on the evaluation of Carcinogenic risk of chemicals to humans." Vol 32. *Polynuclear aromatic compounds.* World Health Organization, Lyon, France.

U.S. Environmental Protection Agency. (1984). "Health effects assessment for polycyclic aromatic hydrocarbons," EPA-540/1-86/013, Washington, DC.

Benzo(k)Fluoranthene
(11,12-Benzo(k)fluoranthene)
Cas No. 207-08-9

Potential sources and exposure

Benzo(k)fluoranthene is a PAH. The reader is referred to the general profile on PAHs for exposure information.

Physical and chemical properties

Property	Value
Molecular weight	252.3 g/mol
Water solubility	4.3×10^{-3} mg/L at 25 °C
Vapor pressure	5.0×10^{-7} mm Hg at 20 °C
K_{oc}	5.5×10^{5} mL/g
log K_{ow}	6.06
Henry's Law Constant	3.94×10^{-5} atm-m^3/mol

Toxicity

Although there are no human data that specifically link exposure to benzo(k)fluoranthene to human cancers, benzo(k)fluoranthene is a component of complex mixtures that have been associated with human cancer. These include soot, coke oven omissions, and cigarette smoke (USEPA as cited in IRIS 1992). IARC concluded that there is sufficient evidence for the carcinogenicity of benzo(k)fluoranthene in experimental animals. Benzo(k)fluoranthene has been administered by skin painting, subcutaneous injection, and intrapulmonary injection. USEPA has classified benzo(k)fluoranthene as a probable human carcinogen (B2).

Toxicokinetics

Like other PAH compounds, benzo(k)fluoranthene is oxidized by liver enzymes to form water-soluble derivatives that can be excreted in urine. No information was available regarding DNA adduct formation or absorption factors. Because of their high lipid solubility, PAHs are believed to be distributed throughout the body. Relative to other tissues, they tend to localize in body fat and fatty tissues.

Ecological effects

The reader is requested to review the toxicity profile for the PAHs for information regarding ecological effects.

References

Integrated Risk Information System (IRIS) on-line database. (1992).

Benzo(a)pyrene
(Benzo(d,e,f) chrysene, 3,4-Benzopyrene, 6,7-Benzopyrene)
Cas No. 50-32-8

Potential sources and exposure

Benzo(a)pyrene (B(a)P) is a PAH. The reader should refer to the general profile on PAHs for exposure information.

Physical and chemical properties

Property	Value
Molecular weight	252.3 g/mol
Water solubility	1.2×10^{-3} mg/L at 20 °C
Vapor pressure	5.60×10^{-9} mm Hg at 25 °C
K_{oc}	5,500,000 mL/g
log K_{ow}	6.06
Henry's Law Constant	1.55×10^{-6} atm-m^3/mol

Toxicity

Lung and skin tumors have been induced in humans by mixtures of PAHs known to contain benzo(a)pyrene (cigarette smoke, roofing tar, and coke oven emissions). It is not possible, however, to conclude from this information that benzo(a)pyrene is the responsible agent (IRIS 1992).

Benzo(a)pyrene is a complete carcinogen when applied to the skin of mice, rats, and rabbits (IARC 1983). Subcutaneous or intramuscular benzo(a)pyrene injection has resulted in local tumors in mice, rats, guinea pigs, monkeys, and hamsters (IARC 1973). Intratracheal instillation of benzo(a)pyrene products produced increased incidences of respiratory tract neoplasms in both male and female Syrian hamsters (IRIS 1992).

Benzo(a)pyrene administered orally to rats and hamsters produces stomach tumors. Dietary benzo(a)pyrene was administered in a subchronic study to male and female CFW-Swiss mice. Stomach tumors were observed in mice consuming 20 or more mg/kg benzo(a)pyrene. Incidence was apparently related both to the dose and the number of administered doses.

Hamsters were chronically exposed to B(a)P by inhalation (IRIS 1992) and were shown to develop respiratory tract tumors. Those hamsters in the highest dose group developed upper digestive tract tumors.

USEPA has classified B(a)P as a Group B2, or probable human carcinogen. The oral cancer slope factor is based on a dietary study in mice.

Toxicokinetics

There are no toxicokinetic data for B(a)P in humans (USEPA 1980). Animal data indicate that B(a)P is readily absorbed after exposure by inhalation or oral intake and distributes to many tissues in the body (USEPA 1980). B(a)P in itself is not believed to be carcinogenic, but metabolized by the cytochrome P-450 dependent mixed function oxidase system, often referred to as the aryl hydrocarbon hydroxylase (AHH) system. The metabolism results in a more hydrophilic compound which is easier to excrete, although is carcinogenic. The hepatic metabolic pathway for B(a)P metabolism is readily inducible by exposure to a variety of chemicals, including B(a)P, and is found in most mammalian tissues. It catalyzes the formation of reactive epoxide intermediates as well as the ultimate carcinogenic form of B(a)P: the B(a)P-7,8-diol-9,10-epoxide (USEPA 1982) which is capable of forming covalent bonds with cellular macromolecules such as DNA, RNA, and proteins. This covalent binding and subsequent alteration of structure and function may result in tumor formation.

Because of their high lipid solubility, PAHs are believed to be distributed throughout the body. Relative to other tissues, they tend to localize in body fat and fatty tissues.

Ecological effects

The reader is requested to review the toxicity profile for PAHs for information regarding ecological effects.

References

Integrated Risk Information System (IRIS) on-line database. (1992).

International Agency for Research on Cancer (IARC). (1973). "Certain monographs on the evaluation of carcinogenic risk to humans." *Polycyclic aromatic hydrocarbons and heterocyclic compounds.* Vol 3. World Health Organization, Lyon, France.

International Agency for Research on Cancer (IARC). (1983). "IARC monographs on the evaluation of the carcinogenic risk of chemicals to humans." Volume 32: *Polynuclear aromatic compounds, Part 1, Chemical, environmental and experimental data.* World Health Organization, Lyon, France.

U.S. Environmental Protection Agency. (1980). "Ambient water quality criteria for polycyclic aromatic hydrocarbons," EPA 440/5-80-069, Washington, DC.

U.S. Environmental Protection Agency. (1982). "An exposure and risk assessment for benzo(a)pyrene and other polycyclic aromatic hydrocarbons. Volume IV," Final Draft Report, Washington, DC.

Benzo(ghi)Perylene
(1,12-Benzoperylene)
Cas No. 191-24-2

Potential sources and exposure

Benzo(ghi)perylene is a polycyclic aromatic hydrocarbon (PAH). The reader is referred to the general profile on PAHs for exposure information.

Physical and chemical properties

Property	Value
Molecular weight	276.3 g/mol
Water solubility	7×10^{-4} mg/L at 25 °C
Vapor pressure	1.03×10^{-10} mm Hg at 25 °C
K_{oc}	1.6×10^{6} mg/L
log K_{ow}	6.51
Henry's Law Constant	5.34×10^{-8} atm-m^3/mol

Toxicity

Although there are no human data that specifically link exposure to benzo(ghi)perylene to human cancers, it is found in complex mixtures that have been associated with human cancer. These include soot, coke oven emissions, and cigarette smoke (IRIS 1992).

IARC (1983) and USEPA (IRIS 1992) concluded that the available data are inadequate to evaluate the carcinogenic potential of benzo(ghi)perylene and is classified as a Group D carcinogen by the USEPA based on the lack of human carcinogenicity data and inadequate data from animal bioassays. Based on a study in which benzo(g,h,i) perylene increased lung tumor incidence when implanted into rat lungs, it was reported that the potency of this compound relative to benzo(a)pyrene was 0.022.

Negative tumorgenicity results were obtained for benzo(ghi)perylene in skin painting studies using mice (IRIS 1992). Mutations due to benzo(ghi)perylene were evident in invitro bacterial mutagenicity tests (IARC 1983).

Toxicokinetics

No data are available regarding the formation of carcinogenic metabolites, DNA adduct formation, enzyme induction, or absorption.

Ecological effects

The reader is requested to review the toxicity profile for PAHs for information regarding ecological effects.

References

Integrated Risk Information System (IRIS) on-line database. (1992).

International Agency for Research on Cancer (IARC). (1983). "IARC monographs on the evaluation of the carcinogenic risk of chemicals to humans." *Polynuclear aromatic compounds, Part 1, Chemical, environmental and experimental data,* Vol 32. World Health Organization, Lyon, France.

Cadmium
Cas No. 7440-43-9

Potential exposure

Cadmium (Cd) and cadmium compounds are typically used as a protective coating for other metals; in the production of metal alloys; fluorescent lamps, semiconductors, photocells, and jewelry; and in batteries, nuclear reactors, engraving, and pesticides. Food and cigarette smoke are the largest potential sources of cadmium exposure for the general population. Ingestion and inhalation are primarily routes of exposure for cadmium. Average cadmium levels in foods within the United States range from 2 to 40 ug/kg. The average level of cadmium in cigarettes ranges from 1,000 to 3,000 ug/kg. Workers can be exposed to cadmium via inhalation or dermal contact while soldering or welding metal. Shellfish can be a major source of cadmium and can contain levels from 100 to 1,000 mg/kg.

Cadmium is also a concern in agricultural soils where sewage sludge is used as compost because it is more readily taken up by plants than other metals. The uptake of cadmium from soil by feed crops may result in high levels of cadmium in beef and poultry (especially in the liver and kidneys).

Physical and chemical properties

Property	Value
Molecular weight	112.4

Toxicity

Acute inhalation of cadmium fumes or dust can cause destruction of lung epithelial cells, resulting in pulmonary edema, tracheobronchitis, and pneumonitis. As a result of breathing high cadmium levels, the acute toxicity can range from a slight irritation of the upper respiratory tract to death. High-level acute oral exposure to cadmium irritates the gastrointestinal epithleum causing nausea, vomiting, and abdominal pain. Breathing lower levels of cadmium for long period of time can lead to accumulation of cadmium in the kidneys thus causing severe kidney damage. Heavy smoking has been reported to considerably increase tissue cadmium levels (Agency for Toxic Substances and Disease Registry (ATSDR 1992)). Nonoccupational inhalation exposure to cadmium is unlikely to be excessive enough to cause respiratory effects. However, chronic inhalation exposure at lower levels can lead to decreased pulmonary function and emphysema. Based on epidemiological and animal studies, it appears that cadmium-induced emphysema is related only to cadmium exposure via inhalation (USEPA 1985a).

The lungs and kidneys are the main target organs for cadmium toxicity following intermediate or chronic duration exposure by the inhalation or oral routes. The earliest clinical signs of cadmium poisoning are proteinuria, glucosuria, and aminoaciduria (USEPA 1985a). Cadmium damages the renal tubules and results in an inhibition of tubular reabsorption but rarely results in renal failure (ATSDR 1992). Prolonged exposure to cadmium which causes renal dysfunction can lead to painful and debilitating bone disease after inhalation or oral exposure as a result of the cadmium effect on calcium metabolism (ATSDR 1992).

A toxicokinetic model is available to determine the level of chronic human oral exposure which results in 200 ug Cd/g wet human renal cortex (the highest renal level not associated with significant proteinuria, the NOAEL). The model assumes a 2.5 percent absorption of Cd from food or 5 percent from water, and that 0.01 percent of the Cd body burden is eliminated per day (USEPA 1985b). The model predicts that the NOAEL for chronic Cd exposure is 0.005 and 0.01 mg Cd/kg/day from water and food, respectively. Thus, based on an estimated NOAEL of 0.005 mg Cd/kg/day for Cd in drinking water, an oral RfD of 0.0005 mg Cd/kg/day (water) was calculated; an equivalent oral RfD for Cd in food is 0.001 mg Cd/kg/day. A risk assessment for an inhalation RfD for cadmium is under review by a USEPA work group.

USEPA has classified cadmium as a Group B1 or probable human carcinogen. This classification is based on occupational epidemiology studies that have shown an increased risk of lung cancer in workers exposed to cadmium via inhalation. A two-fold excess risk of lung cancer was observed in cadmium smelter workers (USEPA 1985b). USEPA has estimated a cancer potency factor (CPF) of 6.1 $(mg/kg/day)^{-1}$ through inhalation route only. The CPF is based on several animal studies (Takenaka et al. 1983; Sanders and Mahaffey 1984).

Toxicokinetics

Cadmium compounds are poorly absorbed from the skin and intestinal tract but are relatively well absorbed from the respiratory tract. Following ingestion or inhalation, cadmium is distributed to most tissues of the body. Initially, highest levels are found in the liver. Later, relocation occurs and highest concentrations appear in the renal cortex (ATSDR 1992). In a study exposing rats daily to cadmium fumes, the distribution of Cd in the tissue was kidney > lung > liver > spleen > aorta > blood (ATSDR 1992). Blood levels in the exposed animals were no different from those of unexposed animals. Similar distributions were found using guinea pigs and monkeys.

Following oral administration, 1 to 5 percent of the dose is absorbed. Variations in absorption are induced by many factors such as age, dietary calcium, and dietary protein levels. Excretion occurs primarily via the kidney at a very slow rate. The biological half-life of cadmium is estimated to be on the order of decades in humans (ATSDR 1992).

Ecological effects

Aquatic and terrestrial organisms bioaccumulate cadmium. Cadmium bioconcentrates in freshwater and marine animals to concentrations hundreds to thousands times higher than the cadmium concentrations in the water.

Levels of cadmium in plant tissue which are considered to be phytotoxic range from 5 to 700 ppm (Chaney 1982), 5 to 30 ppm (Kabata-Pendias and Pendias 1984) and 8 to 15 ppm (Davis, Beckett, and Wollan 1978). It was established that a maximum dietary cadmium concentration chronically tolerated by livestock of 0.5 ppm (based upon cadmium residues in animal products used in human foods).

Aquatic and terrestrial organisms bioaccumulate cadmium. Cadmium bioconcentrates in freshwater and marine animals to concentrations hundreds to thousands times higher than the cadmium concentration in the water.

Of the 44 freshwater genera for which genus mean acute toxicity values are available (USEPA 1984), the most sensitive genus, Salmo, trout is 3,400 times more sensitive than the most resistant genus, Carassius goldfish. Of the freshwater species, rainbow and brown trout appear to be extremely sensitive to cadmium when acutely exposed to concentrations ranging from 1 ug/l to 4 ug/l. The freshwater final acute value of 3.589 ug/l at hardness of 50 mg/l is used to protect against Salmo gairdneri, rainbow trout. However, brown trout is more sensitive than rainbow trout based on an EC50 of 1.63 ug/l from a static test. Chronic mean values derived from acute toxicity values representing 44 genus were used to calculate a final freshwater chronic value of 0.6582 ug/l at hardness of 50 mg/l. The genus mean chronic values for Moina and Daphnia, both cladocerans are below the final freshwater chronic value.

Growth reduction is a major factor toxic effect observed with freshwater aquatic plants and reported values are in the range of concentrations causing chronic effects on aquatic animals. In addition, the lowest toxicity values for freshwater fish and invertebrates species are lower than the lowest values for aquatic plants.

The acute toxicity of cadmium generally increases as salinity increases. The acute values for saltwater invertebrates species range from 41.29 ug/l to 135,000 ug/l for an oligochaete worm. Saltwater mollusks have species mean acute values from 227.9 ug/l for the Pacific oyster to 19,170 ug/l for the mud snail. Saltwater fish species were generally more resistant to cadmium than freshwater fish species with acute values ranging from 779.8 ug/l for the Atlantic silverside to 50,570 ug/l for the mummichog. Of the 33 saltwater genera for which acute values are available, the most sensitive, mysidoposis is 2,000 times more sensitive than the most resistant, Monopylephorus, oligoclaete worms. The saltwater final acute value is 85.09 ug/l and is slightly above the species mean acute value of 78 ug/l for the American Lobster. For the two saltwater species (mysids) for which both chronic and acute toxicity ratios exist, a final saltwater chronic value of 9.345 ug/l was obtained.

Concentrations causing 50 percent reductions in the growth rates of marine diatoms range from 60 ug/l to 175 ug/l. One of the most sensitive marine plants is a red algae, *Champia parvula,* due to growth inhibition at cadmium concentration of 22.8 ug/l.

Bioconcentration factors (BCF) determined with a variety of saltwater invertebrates range from 5 to 3,160. BCF for bivalve mollusks were above 1,000 in long exposures with no indication that a steady state had been reached.

References

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U.S. Environmental Protection Agency. (1985b). "Drinking water criteria document on cadmium," Office of Drinking Water, Washington, DC.

Chromium VI
(Chromium Hexavalent, chromium VI ion, Cr6+)
Cas No. 7440-47-3

Potential sources and exposure

Hexavalent chromium rarely occurs naturally because it is readily reduced to trivalent form in the presence of organic matter. Chromium (VI) is generally produced by industrial processes. Chromium (III) and (VI) compounds are produced by the chemical industry and are used for chrome plating, dye and pigment manufacturing, leather tanning, wood preservatives, and cooling water treatment. Chromium metal is found in asbestos and automotive catalytic converters. Untreated wastewater discharges from electroplating, leather tanning, and textile plants typically contain chromium. For the general population, the common routes of exposure to chromium are inhalation and ingestion of drinking water and food. The wearing down of asbestos brake linings and the exhaust vapors from automobiles, incineration of municipal and sewage sludge, and emissions from cooling towers that use chromium as rust inhibitors contribute to the inhalation exposure pathway.

Physical and chemical properties

Property	Value
Molecular weight	52 g/mol

Toxicity

Unlike chromium III, chromium (VI) is not an essential element. Chronic oral exposures to chromium (VI) typically do not result in toxicity, since the chromium is efficiently reduced to chromium (III) in the gastrointestinal tract.

Dermal exposure to chromium (VI) has been demonstrated to produce irritant and allergic contact dermatitis (IRIS 1998). Primary irritant dermatitis is due to the cytotoxic effect of chromium VI, while the allergic contact dermatitis is due to a two-step cell-mediated immune response. In the first step, chromium is absorbed and triggers an immune response called sensitization. In sensitized individuals, subsequent exposures to threshold levels of chromium will result in allergic contact dermatitis characterized by swelling, papules, redness, dryness, scaling, and cracking of skin. Sensitization may lead to asthmatic attacks following subsequent exposure.

Epidemiological studies have shown that workers employed in chromate production facilities have increased incidences of lung cancer, nasal irritation, atrophy, and nasal septum perforation as well as upper and lower respiratory effects. Chromium-exposed workers are exposed to both the chromium (III) and (VI) compound, but only chromium (VI) has been found to be carcinogenic

according to epidemiological evidence. The USEPA has concluded that only chromium (VI) is carcinogenic. As a result, chromium (VI) compounds are classified as human carcinogens via inhalation (IRIS 1998). The USEPA points out the uncertainty in the relevance of occupational exposure to chromate mists and environmental exposures to chromium particulates (USEPA 1980).

Toxicokinetics

Gastrointestinal absorption of chromium (VI) occurs with greater efficiency than absorption of chromium (III), though absorption of ingested chromium (VI) is estimated to be less than 5percent (USEPA 1998). The absorption of chromium by the lung is dependent upon many factors including the size, oxidation state, solubility of the chromium particles, as well as the activity of alveolar macrophages and the interaction of chromium with reducing agents in the lung. Absorption also occurs through the skin with diffusion constants reported to be 314×10^{-6} cm^2/min (Mali 1963 as cited in USEPA 1998). Factors influencing dermal absorption include the chromium salt employed, the valence state (III or VI), anionic form, concentration, and pH (USEPA 1998).

Once absorbed, chromium (VI) crosses the red blood cell membrane where it can bind to cellular compounds or undergo reduction to chromium (III). There appears to be significant in vivo conversion of chromium (VI) to chromium (III). Chromium (VI) is cleared slowly from blood and rapidly from tissues while the opposite applies to chromium (III). Chromium is distributed primarily to the liver, spleen, bone marrow, lung, and kidney.

Excretion primarily occurs through the urine (50 to 60 percent) with some fecal elimination (about 8 percent) (USEPA 1998). The remainder is deposited in various tissue compartments and has a long biological half-life. Chromium (VI) is eliminated much faster than chromium (III). Adipose and muscle tissue retain chromium for about 2 weeks, while liver and spleen tissue retain chromium for about 1 year.

Ecological effects

USEPA (1980) summarizes studies on the acute effects of hexavalent chromium on various marine species. The species represent a wide range of taxonomic categories and trophic levels and include. The acute value for polychaete worms ranged from 2,000 ug/l (Eisler and Hennekey 1977) to 7,500 ug/l (Reish and Carr 1978). Mollusks displayed relatively high acute values which ranged from 22,000 ug/l for the brackish water clam (Olsen and Harel 1973) to 105,000 ug/l for the mud snail (Eisler and Hennekey 1977). Acute values for fish species ranged from 15,000 ug/l for Atlantic silverside to 91,000 ug/l for mummichog. USEPA (1980) indicates that the chronic value for polychaetes from <13 to 37 ug/l and for mysids it is 132 ug/l. They also indicate that the toxicity to macroalgae ranged from 1,000 to 5,000 ug/l.

Acute toxicity values for chromium (VI) are available for freshwater animal species in 27 genera and range from 23.07 ug/l for a cladoceran to 1,870,000 ug/l

for a stonefly. The few data that are available indicate that the acute toxicity of chromium (VI) decreases as hardness and pH increase. The chronic value for rainbow and brook trout is 264.6 ug/l, and for fathead minnow it is 1.987 ug/l. In all three fishes, a temporary reduction in growth occurred at low concentrations. Six chronic tests with five species of daphnia have chronic values that range from <2.5 to 40 ug/l. Growth of chinook salmon was reduced at 16 ug/l. Green algae are quite sensitive to chromium (VI).

The ambient water quality criteria for chromium (VI) is dependent upon the pH and hardness of the water (Federal Register 1998).

References

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Chrysene
Cas No. 218-01-9

Potential sources and exposure

Chrysene is a (PAH). The reader is referred to the general profile on PAHs for exposure information.

Physical and chemical properties

Property	Value
Molecular weight	228.3 g/mol
Water solubility	1.8×10^{-3} mg/l at 25 °C
Vapor pressure	6.3×10^{-9} mm Hg at 25 °C
K_{oc}	2.0×10^{5} ml/g
log K_{ow}	5.61
Henry's Law Constant	1.05×10^{-6} atm-m^3/mol

Toxicity

Although there are no human data that specifically link exposure to chrysene to human cancers, chrysene is a component of mixtures that have been associated with human cancers. These include coal tar, soots, coke oven emissions, and cigarette smoke (IARC 1983). USEPA has classified chrysene as a Group B2, or probable human carcinogen, on the basis of evidence of carcinogenicity from mouse skin painting and intraperitoneal chrysene injections in male mice which caused an increased incidence of liver tumors. In mouse skin painting assays, chrysene tested positive in both initiation and complete carcinogen studies. The relative tumorigenic potency of chrysene was compared with the potencies of five other polycyclic aromatic hydrocarbons in mouse skin painting assays tested using similar protocols (USEPA 1984). The ranking was as follows: benzo(a)pyrene > dibenz(a,h)anthracene > benzo(b)fluoranthene > benz(a)anthracene > indeno(1,2,3-cd)pyrene > chrysene.

There is limited evidence that chrysene is mutagenic in short-term assays (IARC 1983). There are no experimental data on the teratogenicity of chrysene in mammals. There is no information on the potential effects of chrysene on other endpoints of toxicity.

It was estimated that chrysene had a relative potency to B(a)P of approximately 0.0044. This number can be used in the relative potency method to estimate a cancer potency factor.

Toxicokinetics

Like other PAH compounds, chrysene is oxidized by liver enzymes to form water-soluble derivatives that can be excreted in urine. No information is available regarding dermal or oral absorption coefficients. Because of their high lipid solubility, PAHs are believed to be distributed throughout the body. Relative to other tissues, they tend to localize in body fat and fatty tissues.

Several monohydroxyl and dihydrodiol derivatives of chrysene have been reported (IARC 1983). Epoxides of the 1,2-dihydrodiol and 3,4-dihydrodiol have also been reported (IARC 1983). The 1,2-dihydrodiol and 1,2-diol-3,4-epoxide have been shown to be mutagenic in bacterial and mammalian cells (IARC 1983) and inducers of pulmonary adenomas in newborn mice (IARC 1983). In addition, the 1,2-dihydrodiol has been shown to be a tumor initiating agent on mouse skin (1983). The 1,2-diol-3,4-epoxide is believed to be the metabolite of chrysene that forms adducts with DNA (IARC 1983).

Ecological effects

The reader is requested to review the toxicity profile for PAHs for information regarding ecological effects.

References

International Agency for Research on Cancer (IARC). (1983). "IARC monographs on the evaluation of the carcinogenic risk of chemicals to humans." *Polynuclear aromatic compounds, Part 1, Chemical, environmental and experimental data,* Vol 32. World Health Organization, Lyon, France.

U.S. Environmental Protection Agency. (1984). "Health effects assessment for polycyclic aromatic hydrocarbons," EPA-540/1-86/013, Cincinnati, OH.

Copper
Cas No. 7440-50-8

Potential sources and exposure

Metallic copper (Cu) is used for wires due to its conductive properties and copper compounds are used as insecticides, algicides, and molluscicides, as well as for electroplating reagents. Copper tends to form complexes with both organic and inorganic ligands, such as soils. Copper is used in water distribution piping, cooking utensils, coinage, and natural gas piping. Exposure to copper for the general population is typically via ingestion of drinking water which has passed through copper piping. Occupational exposure to copper occurs primarily through inhalation of fumes or dusts generated during welding.

Physical and chemical properties

Property	Value
Molecular weight	63.5 g/mol

Toxicity

Various effects from acute/subchronic exposures of humans to ingested copper/copper sulfate have been reported: Nausea, vomiting, epigastric pain, headache, dizziness, and abdominal cramps. Dermal exposure to relatively high doses of copper salts may produce skin irritation and eczema. In eyes, copper salts may cause conjunctivitis, and even ulceration and turbidity of the cornea. Inhalation of copper fumes and dust may cause irritation of upper respiratory tract, metallic taste in the mouth, nausea, metal fume fever and in some instances, discoloration of skin and hair. The inhalation of dusts and mists of copper salts through occupational exposure may result in irritation of the nasal mucous membranes and the pharynx, and ulceration and perforation of the nasal septum. No adverse effects via the occupational exposure of copper welders to copper fumes were reported at concentrations up to 0.4 mg Cu/m^3.

Chronic copper toxicity occurs in humans with Wilson's disease, a genetic condition of copper metabolism. Patients with this condition are unable to adequately metabolize copper at normal exposure level, resulting in damage to erythrocytes, kidneys, corneas, and the central nervous system.

Chronic exposure (3 to 15 years) to copper sulfate by vineyard sprayers is reported to have resulted in copper-containing benign granulomas in the lungs.

Toxicokinetics

Copper may be absorbed by dermal, oral, or inhalation exposure routes. Copper absorption is influenced by climate, soil chemistry, diet, water softness,

and pH. Bioaccumulation in biological organisms does not tend to occur upon repeated exposure indicating fairly rapid excretion.

Ecological effects

The toxicity of copper to aquatic life is related primarily to the presence of the free cupric ion, Cu^{2+}, and possibly some of the hydroxy complexes (USEPA 1984). The Cu^{2+} forms stable complexes and precipitates with many inorganic and organic constituents in natural waters. Generally, the concentration of free ion is low compared to total copper present in the water. Organic and inorganic copper complexes appear to be less toxic than the free cupric ion. Aquatic toxicity studies indicate that increasing alkalinity, hardness, and total organic carbon in natural waters decreases copper toxicity. Three major classes of compounds contribute to alkalinity in natural waters. These classes include hydroxide, carbonates and bicarbonates. More copper is complexed as carbonate species, resulting in a significant reduction of the free Cu^{2+}. A change in ionic strength of water alters sensitivity of some aquatic species to copper. The copper ion is significantly more toxic in lower ionic strength waters such as tap water (USEPA 1984).

Acute toxicity data are available for species in 41 genera of freshwater animals. At a hardness of 50 mg/L, the genera range in sensitivity from 16.74 ug/L for Pytochocheilus (northern squawfish) to 10,240 ug/L for Acroneuria (stonefly). The next most sensitive species after Pytchocheilus were the Cladoceran and amphipod species (USEPA 1984). Data for eight species indicate that acute toxicity decreases as hardness increases. Additional data for several species indicate that toxicity also decreases with increases in alkalinity and total organic carbon.

Chronic values are available for 15 freshwater species and range from 3.873 ug/L for brook trout to 60.36 ug/L for northern pike (USEPA 1984). Fish and invertebrate species seem to be about equally sensitive to the chronic toxicity of copper.

The acute sensitivities of saltwater animals to copper range from 5.9 ug/L for the blue mussel to 600 ug/L for the green crab. Chronic tests in a mysid observed adverse effects at 77 ug/L but not at 38 ug/L, yielding an acute-chronic ratio of 3.346 (USEPA 1984). Effects were observed in several saltwater algal species between 5 and 100 ug/L. Oysters can bioaccumulate copper up to 28,000 times and become bluish-green, apparently without significant mortality. In long-term exposures, the bay scallop was killed at 5 ug/L.

References

U.S. Environmental Protection Agency. (1984). "Ambient water quality criteria for Copper," EPA 440/5-84-031, Office of Water Regulations and Standards, Washington, DC.

Dibenzo(a,h)Anthracene (1,2,5,6-Dibenzanthracene, Dibenz(a,h)anthracene) Cas No. 53-70-3

Potential sources and exposure

Dibenzo(a,h)anthracene is a PAH. The reader is referred to the general profile on PAHs for exposure information.

Physical and chemical properties

Property	Value
Molecular weight	278.36 g/mol
Water solubility	5.0×10^{-4} mg/L at 25 °C
Vapor pressure	1.0×10^{-10} mm Hg at 20 °C
K_{oc}	3.3×10^{6} mL/g
log K_{ow}	6.8
Henry's Law Constant	7.30×10^{-8} atm-m^3/mol

Toxicity

Although there are no human data that specifically link exposure to dibenzo[a,h]anthracene with human cancers, dibenzo[a]anthracene is a component of mixtures that have been associated with human cancer. These include coal tar, soots, coke oven emissions, and cigarette smoke (USEPA 1984, IARC 1983).

Dibenzo(a,h)anthracene [DB(a,h)A] has been tested for carcinogenicity in a variety of test species employing a number of different routes of exposure with positive results having been reported in the majority of studies. Little data were identified concerning toxic effects other than tumor induction in the various test species. USEPA has classified dibenzo(a)anthracene as group B2; probable human carcinogen, based on sufficient data from animal bioassays. Dibenzo[a,h]anthracene produced carcinomas in mice following oral or dermal exposure and injection site tumors in several species following subcutaneous or intramuscular administration. Dibenzo[a,h]anthracene and some of its metabolites have induced DNA damage and gene mutations in bacteria as well as gene mutations and transformation in several types of mammalian cell cultures.

Toxicokinetics

Like other PAH compounds, DB(a,h)A is oxidized by liver enzymes to form water-soluble derivatives that can be excreted in urine. No information is available regarding dermal or oral absorption coefficients.

No quantitative data were located concerning the absorption of DB(a,h)A in experimental animals. The 5,6-oxide and the 1,2- 3,4- and 5,6-dihydrodiols have been detected as metabolites of DB(a,h)A after incubation in rat liver

preparations (IARC 1983) and mouse skin in organ culture (IARC 1983). The 5,6-oxide was found to bind to cellular macromolecules in mammalian cells (IARC 1983). Nucleoside adducts have been detected in mouse skin following topical application of DB(a,h)A but were not characterized (IARC 1983).

No information on the tissue distribution or excretion of DB(a,H)A could be located. Because of their high lipid solubility, PAHs are believed to be distributed throughout the body. Relative to other tissues, they tend to localize in body fat and fatty tissues.

Ecological effects

The reader is requested to review the toxicity profile for the PAHs for information regarding ecological effects.

References

International Agency for Research on Cancer (IARC). (1983). "IARC monographs on the evaluation of the carcinogenic risk of chemicals to humans," *Polynuclear aromatic compounds, Part 1, Chemical, environmental and experimental data,* Volume 32. World Health Organization, Lyon, France.

U.S. Environmental Protection Agency. (1984). "Health effects assessment for polycyclic aromatic hydrocarbons," EPA-540/1-86/013, Cincinnati, OH.

Dichlorinated Benzenes (1,2-dichlorobenzene, 1,3-dichlorobenzene, 1,4-dichlorobenzene) Cas No. 106-46-7, 541-73-1, 395-50-1

Potential sources and exposures

The major route of human exposure to the dichlorobenzenes is inhalation of indoor and outdoor air. These compounds are used as room fresheners, moth repellents, fumigants, and cleaners.

Physical and chemical properties (for 1,4-dichlorobenzene)

Property	Value
Molecular weight	147.01 g/mole
Water solubility	79 mg/l at 25 °C
Vapor pressure	1.76 mm Hg at 25 °C
log K_{ow}	3.52
Henry's Law Constant	1.5×10^{-3} atm-m^3/mol at 20 °C

Toxicity

Short-term inhalation exposures to high concentrations of the dichlorobenzenes in humans may result in depression of the central nervous system. The major toxicological effects of inhalation of the dichlorobenzenes are injury to the liver and kidneys. However, it is highly unlikely that exposure concentrations to the general public are high enough to elicit these effects.

The oral RfD for 1,2-dichlorobenzene is based on the NOAEL and LOAEL achieved in chronic and subchronic studies in which rats and mice were given the compound by oral gavage. 1,2-dichlorobenzene is classified as a group D carcinogen.

1,4-dichlorobenzene has caused renal tumors in mice and is presently classified as a group B2 carcinogen. The cancer potency factor that was derived from this study is under review by USEPA Health Effect Assessment Summary Tables (HEAST) (1992) and has not been included in the IRIS database.

Toxicokinetics

Quantitative studies on the absorption of the dichlorobenzenes are unavailable. However, available data on 1,4-dichlorobenzene itself show that about 20 percent was absorbed via inhalation during a 3-hr exposure period

(ATSDR 1992). Since these compounds are structurally similar to benzene, it is thus assumed that 100 percent is absorbed when administered orally. Once absorbed, these compounds tend to accumulate in adipose tissue. The dichlorobenzenes are primarily eliminated in the urine following conjugation in the liver.

References

Agency for Toxic Substances and Disease Registry. (1992). "Toxicological profile for styrene," Draft, U.S. Public Health Service, Washington, DC.

U.S. Environmental Protection Agency. (1992). "Health effects assessment summary tables," OERR 9200.6-303 (92-1), Office of Research and Development, Washington, DC.

Dioxinsfurans

Potential exposure

Dioxins and furans include two classes of halogenated aromatic hydrocarbons, or congeners. Furans are often referred to as "dioxin-like compounds" because their structure and toxicity are similar to dioxins. Dioxin-like compounds are by-products of chlorination processes, for example chlorine bleaching in pulp and paper mills. These compounds are also products of combustion of chlorinated precursor compounds. Dioxins and furans are persistent in the environment and tend to bioaccumulate in the food chain. Ecological receptors are often exposed to mixtures of these compounds in food, soil, and water. Humans may be exposed primarily through their diet and dermal absorption from contaminated ash, soil, and dust.

Physical and chemical properties of 2,3,7,8-TCDD

Property	Value
Molecular weight	322 g/mol
Water solubility	19.3 ng/L
Vapor pressure	7.4×10^{-10} mm Hg at 25 ^{0}C
K_{oc}	$1.15 \times 10_3$ to 3.8×10^7
log K_{ow}	6.64
Henry's Law Constant	1.62×10^{-5} atm-m^3/mol

Toxicity

Exposure to dioxins and furans has been shown to cause acute toxicity to the liver in rodents and rabbits and the thymus in guinea pigs. Epidermal effects, such as chloracne, have been seen in subchronic studies with rodents and monkeys. Other effects due to chronic exposure to dioxin-like compounds are wasting syndrome, hepatotoxicity, enzyme induction, and endocrine effects. In general, congeners without lateral substitution of chlorines and with greater number of chlorine substitutions are more toxic than other congeners.

There is evidence from animal and epidemiological studies that dioxins are furans are immunotoxic. These compounds have also been found to cause developmental and reproductive toxicity in animals and humans. For example, in the Yusho and Yu-Cheng poisoning episodes, the following developmental effects occurred in babies born to mothers who consumed rice oil contaminated with furans and other dioxin-like congeners: fetal death, growth retardation,

structural malformation, organ system dysfunction, and ectodermal dysplasia syndrome. Dioxin-like compounds have also been found to be genotoxic by activating gene transcription through aryl hydroxylase activity (AHA). TCDD, the most potent of all the dioxin congeners has been shown to be a multisite carcinogen in both sexes of mice and in hamsters. It is believed that there are multiple mechanisms for TCDD's "tumor promoting" activity. The carcinogenic effects of TCDD are hepatocellular carcinomas and hepatocellular hyperplastic nodules.

Toxicokinetics

Following oral exposure, gastrointestinal absorption of TCDD in animal studies is nonlinear, with the greatest absorption occurring at ≤ 0.01 u mol/kg. Gastrointestinal absorption of TCDF in animals is almost complete (90 percent or greater). In humans, absorption via oral exposure is variable, incomplete, and congener- and vehicle-specific. Transpulmonary absorption is similar to that observed after oral exposure, however, the rate of absorption via dermal routes is slower. Dioxin-like compounds are often associated with lipoprotein in the blood and in lymph, thus they may be distributed to organs of the body in proportion to the amount of blood flow to each organ and organ size. The adrenal glands and muscle are the first organs to which dioxins and furans are distributed, followed by the liver, adipose tissue and skin. The highest concentrations of dioxin-like compounds have been found in the liver and adipose tissue. Dioxins and furans are metabolized by the body to polar compounds and excreted as urine, bile, and feces.

Ecological effects

Early life stages of animals have been more sensitive to TCDD than adult animals. Studies have shown that TCDD is directly toxic to pike, rainbow trout, lake trout, and Japanese medaka. The toxic effects on young fry of these fish species are edema, hemorrhage, arrested growth and development, and death. TCDD has been extremely toxic to bird eggs. Signs of toxicity are species-specific; however, embryo mortality is common to all species.

Fluorene
Cas No. 86-73-7

Potential sources and exposure

Fluorene is a (PAH). The reader should refer to the general profile on PAHs for exposure information.

Physical and chemical properties

Property	Value
Molecular weight	166.7g/mol
Water solubility	1.69 mg/L at 25 °C
Vapor pressure	7.1×10^{-4} mm Hg
K_{oc}	7,300 mL/g
log K_{ow}	4.2
Henry's Law Constant	6.4×10^{-5} atm-m^3 /mole

Toxicity

Due to the lack of data on the toxicity of fluorene to humans, IARC (1983) concluded that the available data in experimental animals was inadequate to permit an evaluation of the carcinogenicity of fluorene. The USEPA's Carcinogen Assessment Group has classified fluorene in Group D: Not classifiable as human carcinogen (IRIS 1992).

The RfD for oral exposure to fluorene is 0.04 mg/kg-day, based on subchronic exposure to flluorene in mice by oral gavage. The LOAEL is 250 mg/kg-day based on hematological effects; the NOAEL is 125 mg/kg-day.

Toxicokinetics

Like other PAH compounds, fluorene is oxidized by liver enzymes to form water-soluble derivatives that can be excreted in urine. No information is available regarding dermal or oral absorption coefficients.

Due to their high lipid solubility, PAHs are believed to be distributed throughout the body. Relative to other tissues, they tend to localize in body fat and fatty tissues. Elimination of PAHs is primarily via the hepatobiliary tract.

Ecological effects

The reader is requested to review the toxicity profile for the PAHs for information regarding ecological effects.

References

Integrated Risk Information System (IRIS) on-line database. (1992).

International Agency for Research on Cancer (IARC). (1983). "IARC monographs on the evaluation of the carcinogenic risk of chemicals to humans," *Polynuclear aromatic compounds, Part 1, Chemical, environmental and experimental data,* Volume 32. World Health Organization, Lyon, France.

Fluoranthene
(Idryl; 1,2-(1,8-Naphthylene)benzene; Benzo(jk)fluorene)
Cas No. 206-44-0

Potential sources and exposure

Fluoranthene is a PAH. The reader is referred to the general profile on PAHs for exposure information.

Physical and chemical properties

Property	Value
Molecular weight	202.30 g/mol
Water solubility	0.206 mg/L at 25 °C
Vapor pressure	5.0×10^{-6} mm Hg
K_{oc}	3.8×10^{4} mL/g
log K_{ow}	4.9
Henry's Law Constant	6.5×10^{-6} atm-m^3/mol

Toxicity

Fluoranthene has been tested for carcinogenicity, with negative results, in several tests including skin painting studies (as cited in IARC 1983) and a subcutaneous injection study (as cited in IARC 1983). USEPA has not classified fluoranthene with regard to its carcinogenicity due to inadequate evidence (IRIS 1992). However, equivocal evidence for mutagenicity of fluoranthene in short-term bacterial and mammalian tests has been reported (IRIS 1992).

The RfD for oral exposure to fluoranthene is 0.04 mg/kg-day, based on a study in mice in which subchronic exposure by gavage was associated with kidney toxicity, increased liver weights and alterations in blood characteristics (IRIS 1992).

A study of fluoranthene's developmental toxicity was performed in which intraperitoneal injection to pregnant mice resulted in an increased rate of fetal resorption (IRIS 1992).

Toxicokinetics

Like other PAH compounds, fluoranthene is oxidized by liver enzymes to form water-soluble derivatives that can be excreted in urine. No information is

available regarding dermal or oral absorption coefficients. Because of their high lipid solubility, PAHs are believed to be distributed throughout the body. Relative to other tissues, they tend to localize in body fat and fatty tissues.

LaVoie and coworkers (1982 as cited in IARC 1983) detected the 2,3-dihydrodiol metabolite of fluoranthene which is mutagenic in bacterial tests with an exogenous activation system.

Ecological effects

The reader is requested to review the toxicity profile for the PAHs for information regarding ecological effects.

References

Integrated Risk Information System (IRIS) on-line database. (1992).

International Agency for Research on Cancer (IARC). (1983). "IARC monographs on the evaluation of the carcinogenic risk of chemicals to humans," *Polynuclear aromatic compounds, Part 1, Chemical, environmental and experimental data,* Volume 32. World Health Organization, Lyon, France.

Indeno(1,2,3-cd)Pyrene (2,3-Phenylenepyrene; 2,3-o-Phenylenepyrene) Cas No. 193-39-5

Potential sources and exposure

Indeno(1,2,3-cd)pyrene is a PAH. The reader should refer to the general profile on PAHs for exposure information.

Physical and chemical properties

Property	Value
Molecular weight	276.34 g/mol
Water solubility	6.20×10^{-1} ppm at 25 °C
Vapor pressure	1.0×10^{-10} mm Hg at 20 °C
K_{oc}	1.6×10^{6} mL/g
log K_{ow}	6.5
Henry's Law Constant	6.86×10^{-8} atm-m^3/mol at 20 °C

Toxicity

Although there are no human data that specifically link exposure to indeno(1,2,3-cd)pyrene to human cancers, indeno(1,2,3-cd)pyrene is a component of mixtures that have been associated with human cancer. USEPA has classified indeno(1,2,3-cd)pyrene as a B2 or probable human carcinogen on the basis of positive results in mice and bacterial mutation assays. Indeno(1,2,3-cd)pyrene has produced tumors in mice following lung implants, subcutaneous injection, and dermal exposure (IRIS 1992).

The relative tumorigenic potency of indeno(1,2,3-cd)pyrene was compared with the potencies of five other polycyclic aromatic hydrocarbons in mouse skin painting assays conducted using similar protocols (USEPA 1984). The ranking was as follows: B(a)P > dibenzo(ah)anthracene > benzo(b)fluoranthene > benzo(a)anthracene > indeno(1,2,3-cd)pyrene > chrysene.

Indeno(1,2,3-cd)pyrene induced mutations bacterial assays in Salmonella typhimurium strain TA 100 at a concentration of 20 ug/plate and in strain TA 98 at a concentration of 2 ug/plate in the presence of an exogenous metabolic activating system (IARC 1983). Due to the equivocal mutagenicity testing data, IARC (1983) considered the available evidence inadequate to classify indeno(1,2,3-cd)pyrene as a mutagen.

Toxicokinetics

There are no toxicokinetic data of indeno(1,2,3-cd)pyrene in man (USEPA 1980). In general, many polycyclic aromatic hydrocarbons (PAHs) can produce toxicity after inhalation, oral, or dermal exposure. Thus, it is believed that they are readily absorbed after exposure by these routes. Because of their high lipid solubility, PAHs are believed to be distributed throughout the body. Relative to other tissues, they tend to localize in body fat and fatty tissues. PAHs are generally metabolized by the microsomal mixed function oxidase system and eliminated primarily via the hepatobiliary tract.

Ecological effects

The reader is requested to review the toxicity profile for the PAHs for information regarding ecological effects.

References

Integrated Risk Information System (IRIS) on-line database. (1992).

International Agency for Research on Cancer (IARC). (1983). "IARC monographs on the evaluation of the carcinogenic risk of chemicals to humans," *Polynuclear aromatic compounds, Part 1, Chemical, environmental and experimental data,* Volume 32. World Health Organization, Lyon, France.

U.S. Environmental Protection Agency. (1980). "Ambient water quality criteria for polycyclic aromatic hydrocarbons," EPA 440/5-80-069, Washington, DC.

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Lead
Cas No. 7439-92-1

Potential sources and exposure

For the general population, exposure to lead (Pb) occurs by eating foods that contain lead, inhalation of outdoor/household dust, incidental ingestion of soil and lead paint, and through the consumption of lead in drinking water. Through atmospheric deposition, lead enters the environment. Lead can be translocated from the soil into plants. Lead may enter prepared foods when food is prepared in improper glazed pottery and ceramic dishes. Drinking water from acidic water supplies may contain lead which enters through the distribution system (lead pipes, solder, and brass faucets). Household dust may contain lead that is attributed to the outdoor lead in soil and the weathering of lead-based paints. Most childhood lead exposures result from inhaling lead paint dust, eating soil or dust that contains lead, and drinking water containing lead.

Physical and chemical properties

Property	Value
Molecular weight	207.2 g/mol

Toxicity

Toxic effects resulting from chronic lead exposures are well documented and many have been associated with particular blood-lead levels. Preschool aged children develop symptoms of lead intoxication at lower blood lead levels than do adults. Lead is particularly harmful to the developing brain and nervous system of young children and fetuses (Center for Disease Central (CDC) 1991). Research has shown that adverse effects of lead on the developing nervous system occur at blood-lead levels as low as 10-15 ug/dL. The recommended target level for blood lead in children is 10 ug/dl. Children with a consistent blood-lead level of 15-19 ug/dL can suffer adverse effects such as mild to moderate decrease in IQ, increase in hearing thresholds, shortened attention span, and learning and behavioral difficulties. Children with blood-lead levels between 20-69 ug/dL are considered "lead poisoned." Depending upon the age of the child, blood-lead level and duration of exposure may exhibit speech delays, hyperactivity, regression of recently acquired skills, irritability, and change in appetite. The gastrointestinal system is one of the earliest to show symptoms of acute lead intoxication with colic (acute abdominal pain) considered a consistent early symptom of lead poisoning. Lead encephalopathy can result from blood-lead levels greater than 100 ug/dL and is characterized by irritability, loss of memory, and ability to concentrate, delirium, hallucinations, cerebral edema, and coma (ATSDR 1992).

Hematologic effects appear to be among the most sensitive indicators of lead absorption. Lead interference with heme synthesis has been noted in humans and other mammalian species at blood levels below 10-15 ug/dL. Lead can also lead to the accumulation of porphyrin in erythrocytes with elevated levels of erythrocyte protoporphyrin (EP) associated with blood lead levels of 25-30 ug/dL in adults and 15 ug/dL in children. Anemia is characteristic of more severe cases of lead poisoning, resulting from erythrocyte destruction and reduced hemoglobin synthesis (ATSDR 1992).

Renal toxicity has also been observed in victims of lead intoxication. Reversible proximal tubule damage has been observed primarily in cases of short-term exposure with reduced glomerular function associated with more chronic exposures (ATSDR 1992). In adults, chronic exposures to lead can result in hypertension. Acute exposures can result in peripheral neuropathy and/or nephropathy. Due to the relationship between maternal body lead stores and fetal circulation, fetal development can be adversely affected by elevated maternal body-lead burdens.

USEPA classifies inorganic lead as a category B2, probable human carcinogen. There is inadequate evidence of carcinogenicity based on human studies, but several animal bioassays have shown statistically significant increases in renal tumors following dietary and drinking water exposure to lead acetate or lead subacetate, two soluble lead salts (IRIS 1999). USEPA has not calculated a cancer slope factor for inorganic lead because of the large uncertainties involved, including the effect of age, health, nutritional status, and body burden (IRIS 1999).

The USEPA has not established a risk reference dose (RfD) for lead because it appears that some of the observed effects occur at such low doses as to be essentially without a threshold (IRIS 1999). Because a USEPA derived reference dose is not available, an alternative approach called the integrated uptake/ biokinetic model is used to evaluate the potential for adverse health effects due to lead. This is a validated model that calculates blood-lead levels based on estimated exposure doses of lead to children in to various media such as food, soil, dust, and water. Once blood lead levels are estimated, adverse effects can be predicted. To determine an estimation of the health risk due to exposure to lead at the site of interest, a threshold based on blood lead has been defined, at 10 ug/dl (CDC 1991).

Toxicokinetics

Absorption through the gastrointestinal tract is a function of many factors including the fasting state and nutritional status of the individual, solubility of the lead, and particle size. For dietary lead, absorption in children is approximately 50 percent compared with 5 to 15 percent gastrointestinal lead absorption in adults (World Health Organization (WHO) 1995). Lead is not well absorbed dermally, from 0.006 percent to less than 0.3 percent (WHO 1995). Lead is well absorbed by the lungs, and absorption depends on a number of factors. These include whether the lead is in particulate or vapor form and the

size distribution of the particles. The lung retains a very minor fraction of particles over 0.5 um in mean maximal external diameter. The larger particles are cleared by the respiratory tract and then swallowed. Those particles less than 0.5 um are efficiently absorbed (WHO 1995).

Distribution in the body occurs in a similar manner regardless of the route of absorption. Lead is distributed to both soft tissue and bone, although distribution is not homogenous. Three pools of lead have been identified: blood, bone, and soft tissues. This compartmentalization and distribution to these compartments forms the basis for the biokinetic models for lead. Blood is the compartment in which lead is most often measured as a marker of recent exposure (due to the short half-life of lead in blood), although lead in blood is also derived from lead stored in tissues. Human bone has at least two, possibly three, kinetically distinct lead compartments with differing abilities to mobilize lead to the blood. Lead in bone may contribute as much as 50 percent of blood lead, so bone itself is a significant source of lead. The fraction of lead in bone increases with the age of the person, therefore this is more of a concern for adults. In the body, about 94 percent of the adult body burden of lead is localized in the skeleton, about 4 percent is in the blood, and 2 percent is in soft tissue. In children, only about 73 percent of lead in the body is in the bone. Mobilization of lead during pregnancy and lactation will elevate blood lead concentrations and can be of concern for fetal exposures. Lead is efficiently transferred across the placental membranes. The lead concentration in human umbilical cord blood is 85 to 90 percent that of maternal blood, and lead accumulation in fetal tissues is proportional to maternal blood lead levels (World Health Organization 1995).

Absorbed lead is eliminated through urinary and fecal excretion. The unabsorbed gastrointestinal lead and the airborne lead that was swallowed are also eliminated in feces. Based on estimates of first-order elimination of half-lives for lead in blood, a constant lead intake rate over the course of months is required to maintain a steady-state blood-lead level. Exposures of 1 day/week are sufficient to maintain these steady-state conditions (USEPA 1994).

Ecological effects

The effects of metals in soils are very much dependent upon the availability of the metal from the soil matrix. Lead seems to be tightly bound by most soils, and substantial amounts must accumulate before it affects the growth of higher plants (Eisler 1988). Plants readily accumulate lead in soils with low pH or low organic content. Lead has very high residence time in forest litter. Estimates range from 220 years to 500 years (as summarized in Eisler 1988). Lead toxicosis has been observed in plants from lead concentrations ranging from 0.005 to 33,000 mg/L. Effects include growth stimulation (at low levels), growth inhibition, leaf yellowing, abscission, inhibition of mitosis and chlorophyll synthesis, loss of turgor pressure and death.

Eisler (1988) reviewed the potential effects of lead contamination to wildlife for the U.S. Fish and Wildlife Service. Lead toxicity in water fowl through the ingestion of lead pellets is well documented. Several accidental lead poisoning

cases have been reported in livestock. Cattle and horses in the vicinity of a lead smelter died due to lead exposure. A sharp decrease in total milk yield and a significant increase in stillbirths and abortions were reported in dairy cattle that ingested lead-contaminated hay. Eisler also notes that there is no evidence for biomagnification of lead in the food chain of vegetation, to cattle, to the dung beetle, nor is there convincing evidence that any terrestrial vegetation is important in food chain biomagnification of lead.

At a water hardness of 50 mg/L, the acute sensitivities of ten freshwater species range from 142.5 ug/L for an amphipod to 235,000 ug/L for a midge (USEPA 1984). The lowest and highest available chronic values (12.26 and 128.1 ug/L) are both for a cladoceran. Freshwater algae are affected by concentrations of lead above 500 ug/L, based on data for four species. Acute values are available for 13 marine fauna and range from 315 ug/L for the mummichog to 27,000 ug/L for the soft-shell clam. A chronic toxicity test was conducted with a mysid; unacceptable effects were observed at 37 ug/L. The ambient water quality criteria for lead is dependent upon the pH and hardness of the water (*Federal Register* 1998).

References

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Integrated Risk Information System (IRIS) on-line database, accessed 1/99.

U.S. Environmental Protection Agency. (1984). "Ambient water quality criteria for lead," Office of Water, Washington, DC.

U.S. Environmental Protection Agency. (1994). "Guidance manual for the integrated exposure uptake biokinetic model for lead in children," EPA 540-R-93-081 (PB93-963510), Office of Emergency and Remedial Response, Washington, DC.

World Health Organization. (1995). "Environmental health criteria for inorganic lead. No.165," World Health Organization, Geneva.

Mercury
Cas No. 7439-97-6

Potential sources and exposure

Mercury (Hg) is an element that can occur naturally in the environment in several forms. Elemental Hg is used in barometers, thermometers, batteries, and paints. Mercury can combine with other chemicals in the environment, such as chlorine, carbon, or oxygen to form "inorganic" or "organic" mercury compounds. Compounds of Hg have been used as fungicides and preservatives. Most human and ecological receptors are exposed to mercuric compounds that have been produced by industrial sources. Human exposure is generally through occupational exposure via inhalation of Hg vapors, dermal contact with mercuric compounds, or nonoccupational ingestion of mercuric compounds in foods such as fish that have high levels of methylmercury in their systems.

Physical and chemical properties

Property	Value
Molecular weight	200.59 g/mol (metallic)
Vapor pressure	0.002 mmHg at 25 °C

Toxicity

The route of exposure and the type of mercuric compound to which the individual is exposed will determine the toxicity. The central nervous system is the target system for Hg toxicity. Following acute exposures, several adverse neurological effects have been noted in humans, including tremors, decreases in motor function, and headaches. These acute effects may be reversible. Elemental mercury is not highly toxic as an acute poison, although inhalation of high concentrations of mercury vapor for relatively short duration can cause bronchitis, chest pains, dyspnea, coughing, salivation, and diarrhea. Mercury compounds are primary skin irritants and may cause dermatitis on contact. Exposures to chronic low doses of the Hg vapor can result in short-term memory deficits, decreased nerve conduction, and visual disturbances. Long-term effects may also include memory loss, hallucination, and mental deterioration.

The reference dose for inhalation of elemental Hg is based on subchronic human studies in which a NOAEL of 0.009 was observed for neurological effects. The oral RfD is based upon a subchronic exposure to rats in which immunological effects were observed at the lowest LOAEL.

The USEPA has classified mercury as a Class D carcinogen based upon the lack of human data and inadequate animal data.

There is some evidence of genotoxicity of Hg based upon an epidemiological study (ATSDR 1989) in which there was a statistical relationship between chromosome breaks and concentrations of methyl mercury in the blood of Swedish subjects on fish diets.

Toxicokinetics

The pharmacokinetics of Hg depend largely on its chemical form. At low doses, most of the elemental Hg is oxidized to the divalent cation which does not cross the blood-brain barrier. Oral absorption of elemental Hg has been estimated to be between 0.01 and 0.1 percent (ATSDR 1989). Dermal absorption of metallic Hg is estimated to be approximately 2 percent, while the absorption efficiency via inhalation is probably closer to 80 percent. Oral absorption efficiency of inorganic Hg is estimated to be approximately 7.5 percent based upon animal and human feeding studies. The oral absorption efficiency of methylmercury is reported to be as high as 95 percent.

Ecological effects

Mercury is recognized as one of the most toxic of the heavy metals. Numerous physical factors can affect the acute and chronic toxicities and bioaccumulation of the various forms of Hg. Data are available on the acute toxicity of Hg to at least 28 genera of freshwater animals. Acute values for water-borne invertebrate species range from 2.1 ug/L for Daphnia to 2,000 ug/L for three insects. Acute values for fishes range from 30 ug/L for the guppy to 1,000 ug/L for some tropical marine organisms. Few data are available for various organomercury compounds, although they appear to be at least five times more acutely toxic than metallic mercury. Available chronic data indicate that methylmercury is the most chronically toxic of the mercury compounds. This is in part because of the ability of methylmercury to bioconcentrate.

References

Agency for Toxic Substances and Disease Registry. (1989). "Toxicological profile for mercury," U.S. Public Health Service, Washington, DC.

Naphthalene
Cas No. 91-20-3

Potential sources and exposures

Naphthalene is PAH. The reader should refer to the general profile on PAHs for exposure information. Naphthalene is found in moth balls; exposure may arise through inhalation and dermal and ingestion routes.

Physical and chemical properties

Property	Value
Molecular weight	128.2 g/mol
Water solubility	31.7 mg/L at 25 °C
Vapor pressure	8.2 x 10-2 mm Hg at 25 °C
K_{oc}	940 mL/g
log K_{ow}	3.3
Henry's Law Constant	4.8×10^{-4} atm-m^3/mole

Toxicity

In humans, exposure to sufficient concentrations of naphthalene through inhalation, ingestion, or dermal contact may cause intravascular hemolysis or the less severe symptoms of eye irritation, headache, confusion, tremors, nausea, vomiting, abdominal pain, and bladder irritation. In severe cases hematological effects have included red cell fragmentation, icterus, severe anemia, leukocytosis and dramatic decreases in hemoglobin, hemacrit, and red cell counts. Hemolysis can also lead to renal disease from precipitated hemoglobin (USEPA 1982). Poisonings have occurred in humans as a result of the ingestion of moth balls as well as from clothing infants in materials that had been stored in moth balls. A study of workers exposed to naphthalene for a period of 5 years found corneal ulceration, cataracts, and some lenticular and general opacities in 8 of the 21 employees examined. No data were located indicating naphthalene to be an hepatic enzyme inducer.

Ecological effects

A variety of aquatic species has been exposed to naphthalene and most acute tests were under static procedures with unmeasured test concentrations. All but two LC50 effect levels for fish and invertebrate species are in the range of 2,300 to 8,900 ug/L. One embryo-larval test with the fathead minnow demonstrated adverse effects at a test concentration of 850 ug/L.

Daphnia magna is the only tested freshwater invertebrate species for which the acute toxicity of naphthalene has been determined (USEPA 1982). The reported 48-hr EC50 is 8,570 ug/L.

Flow-through tests were conducted with measured concentrations for the rainbow trout and the fathead minnow. The trout appeared to be more sensitive with a 96-hr LC50 of 2,300 ug/L. The 96-hr LC50 for the fathead minnow tested at 14 °C degrees centigrade was 4,900 ug/L, at 24 °C the LC50 was 8,900 ug/L. The LC50 of 150,000 ug/L for the mosquitofish appears to be atypical but the result cannot be discounted.

LC50 (96 h) values for the polycheate, Neanthes arenaceodentata, (Pacific oyster), and the grassshrimp are 3,800, 199,000, and 2,350 ug/L, respectively. The 24-hr LC50 values for one fish and two saltwater shrimp species range from 2,400 to 2,600 ug/L.

With the exception of the mosquitofish and the Pacific oyster, all LC50 and EC50 values, regardless of test method, fall within the narrow range of 2,300 to 8,900 ug/L for nine freshwater and saltwater species.

Tests have been conducted to determine the chronic toxicity of naphthalene to ecological receptors. An embryo-larval test has been conducted with the fathead minnow and the resultant chronic value is 620 ug/l. When this concentration is divided by the geometric mean LC50 value of 6,600 ug/L for this species an acute-chronic ratio of 2 is obtained. No other species have been tested under chronic conditions.

There is only one reported test that determined an apparent equilibrium bioconcentration factor for naphthalene. After 9 days, the bioconcentration factor for a copepod was 5,000. Bioconcentration data for other species for exposures of 1 hr to 1 day range from 32 to 77 and indicate that equilibrium does not occur rapidly when those results are compared to the 9 day value of 5,000.

References

U.S. Environmental Protection Agency. (1982). "An exposure and risk assessment for benzo(a)pyrene and other polycyclic aromatic hydrocarbons," Volume IV. Final Draft Report, Washington, DC.

Nickel
Cas No. 744-02-0

Potential sources and exposure

Nickel is a naturally occurring metal that is mined and is combined with other metals to form alloys. Nickel is emitted into the air through fossil fuel combustion, incinerators, chemical and cement manufacturing, coke ovens, and nickel recovery operations. Evidence has accumulated indicating that nickel may be a trace metal essential for human health.

Physical and chemical properties

Property	Value
Molecular weight	58.69 g/mol
Water solubility	insoluble at 25 °C
K_{oc}	No data
log K_{ow}	No data
Henry's Law Constant	No data

Toxicity

The target organs of nickel toxicity are skin and lungs. Allergic contact dermatitis to nickel-containing metals is common in the general public. The major adverse effects seen as a result of high exposure levels to nickel, likely found only in the workplace, include dermatitis, chemical pneumonitis, and lung and nasal cancers. Nickel carbonyl is extremely toxic, resulting in chest pain, dry coughing, cyanosis, gastrointestinal symptoms, sweating, visual impairment, and weakness. This is often followed by pulmonary hemorrhage and edema. Survivors may be left with pulmonary fibrosis.

The USEPA classifies nickel as a Group A - Human Carcinogen based on epidemiological studies in which a causal association exists between exposure to nickel refinery dust and lung and nasal tumors.

Toxicokinetics

Nickel is poorly absorbed from the gastrointestinal tract. Absorption from the respiratory tract is dependent on the solubility of the nickel compounds, with higher urinary nickel observed in workers exposed to soluble nickel compounds (Ni chloride, Ni sulfate) than those exposed to insoluble nickel compounds (Ni oxide, Ni subsulfide). Nickel applied directly to the skin can be absorbed

into the skin where it may remain rather than entering the systemic circulation. Following inhalation exposure, nickel tends to accumulate in the lungs. Nickel can cross the placenta and it can accumulate in breastmilk. Regardless of the exposure route, absorbed nickel is excreted in the urine.

Ecological effects

a. *Bioaccumulation.* Nickel concentrations in plants are generally low, < 150 mg/kg dry weight, but occasional reports will show much higher concentrations of 150 to 700 mg/kg. Likewise, invertebrate concentrations are low, usually < 5 mg/kg. Nickel cannot be considered a significant, widespread contaminant except at certain site-specific points. Uptake in invertebrates occurred principally through the water and ingested particulate nickel was excreted. In fish, concentrations again are generally low, < 0.5 mg/kg wet weight, but instances of higher concentrations do exist near polluted areas (1 to 2 mg/kg wet weight).

b. *Toxic Effects to Aquatic Organisms.* Nickel (Ni^{2+}) is considered moderately to highly toxic to most aquatic plant species. To invertebrates, Ni^{2+} is one of the least toxic inorganic agents. To both marine and freshwater fish, Ni^{2+} is relatively nontoxic but when exposed to low levels over extended periods effects include reduced skeletal calcification and reduced diffusion capacity of gills. Both acute and chronic toxicity of Ni^{2+} is strongly related to water hardness.

Organocarbamate Pesticides

Potential exposure

Organocarbamate pesticides are one of several classes of insecticides, including compounds such as carbaryl, aldicarb, and zectran. The organocarbamates are not broad-spectrum insecticides, and some common household insect pests are relatively immune to the effects of these chemicals. Unlike the organophosphate insecticides, most of the organocarbamate insecticides have low dermal toxicities. However, due to the high toxicity of aldicarb by both the oral and dermal routes, it has restricted use in the United States and is recommended only for limited use in greenhouse operations. Aldicarb is released to soil as a systemic insecticide for soil use. Carbaryl is a widely employed insecticide used against a variety of insect pests of cotton, fruits, vegetables, ornamental trees and shrubs, and animals and livestock. It is also used as a molluscicide. Humans may be exposed to organocarbamate insecticides in contaminated air, soils, water, and food by inhalation, dermal contact, and ingestion exposure routes.

The persistence of organocarbamates in the environment varies with each individual compound and the chemical properties of the surrounding soils and water. The reported persistence of carbaryl ranges from nonpersistent in aerobic conditions, with effectiveness lasting from a few hours to several days, but rarely more than 12 weeks, to moderately persistent, with effectiveness ranging from 1 to 18 months (Briggs and Council 1992). The reported persistence of aldicarb ranges from nonpersistent, with effectiveness lasting from a few hours to several days, but rarely more than 12 weeks, to persistent, retaining toxicity for years (Briggs and Council 1992). Neither of these compounds bind strongly to soil and both have potential to leach to groundwater.

Physical and chemical properties of carbaryl

Property	Value
Molecular weight	201.22 g/mol
Water solubility	32 mg/L at 20 °C
Vapor pressure	1.36×10^{-6} mm Hg at 25 °C
K_{oc}	370 to 390
log K_{ow}	2.36
Henry's Law Constant	1.28×10^{-8} atm-m^3/mol at 20 °C

Toxicity

The mode of action of the organocarbamates, like the organophosphates, is inhibition of acetylcholinesterase. Inhibition of acetylcholinesterase results in accumulation of endogenous acetylcholine, a chemical transmitter of neural impulses in nerve tissue and effector organs. This results in an overactivity of cholinergic components of the autonomic nervous system, inhibition of

conduction across myoneural junctions in skeletal muscle, and interference of CNS synaptic transmission. The signs and symptoms of poisoning, which are the same as those for the organophosphate insecticides, are typically cholinergic with lacrimation, salivation, miosis, convulsion, and death. The associated symptoms mimic the muscarinic, nicotinic, and CNS actions of acetylcholine and the severity of the effects is dose-dependent.

Acute toxicities, represented by the oral LD50 in male rats, ranged from 0.8 mg/kg for aldicarb to 850 mg/kg for carbaryl (Klaassen, Amdur, and Doull 1986). Carbaryl is considered one of the least acutely toxic carbamate insecticides. Long-term dietary exposures to carbaryl in rats resulted in kidney and liver toxicity levels similar to those observed for rat cholinesterase inhibition in a separate chronic study (IRIS 1997).

Carbaryl is teratogenic in several experimental animals with widely varying no-observed effect levels. However, in most species, the doses for effects on fetuses were near the maternal toxic doses. Although the lowest effect levels were observed for dogs (a tenth of the toxic dose to the mother), these studies were judged inappropriate for human health risk assessment because of differences in the metabolism of carbaryl between dogs and humans (IRIS 1997).

Chronic toxicity test results indicate carbaryl as a potential carcinogen and mutagen, while aldicarb has been indicated as a suspect mutagen (Briggs and Council 1992). Carbaryl has not been evaluated by the USEPA for its human carcinogenic potential.

Toxicokinetics

Organophosphates are absorbed by the respiratory tract, mucous membranes, skin, and gastrointestinal tract. The carbamate insecticides are direct inhibitors of acetylcholinesterase and do not require metabolic activation (Klaassen, Amdur, and Doull 1986). Hydrolytic reactions result in metabolites that lack anticholinesterase activity. Various oxidation steps are catalyzed by mixed function oxidases. The products formed by these reactions are not always less toxic than the parent compound (Klaassen, Amdur, and Doull 1986). Unlike the organophosphates, the organocarbamates are reversible inhibitors of cholinesterase, and their duration of action is relatively short. Atropine alone is the recommended antidote for organocarbamate poisoning.

Ecological effects

Bees are extremely sensitive to the organocarbamate insecticides, which can also disrupt schooling behavior of fish, and are considered teratogens in fish (Briggs and Council 1992). Certain organocarbamates are toxic to earthworms and invertebrate populations. Aldicarb is reported to be highly toxic to birds, fish, and aquatic insects, while carbaryl is highly toxic to fish, crustaceans, earthworms, aquatic worms, and aquatic insects (Briggs and Council 1992). Neither aldicarb nor carbaryl are expected to bioconcentrate significantly in aquatic organisms.

References

Briggs, S. A., and Council, Rachel Carson. (1992). *Basic guide to pesticides: Their characteristics and hazards.* Taylor & Francis, Washington, DC.

Integrated Risk Information System (IRIS). (1997). On-line database.

Klaassen, C. D., Amdur, M. O., and Doull, J. (1986). *Casarett and Doull's toxicology: The basic science of poisons.* 3rd ed., Macmillan, New York.

Organochlorine Pesticides

Potential exposure

Organochlorine pesticides are one of several classes of insecticides, which include the chlorinated ethane derivatives (DDT and methoxychlor), the cyclodienes (chlordane, aldrin, dieldrin, heptachlor, endrin, and toxaphene), and the hexachlorocyclohexanes (lindane). During the 1940s to the 1960s, the organochlorine insecticides were used extensively in agriculture, soil, and structure insect control, as well as in malaria control programs. However, due to their long-term persistence in the environment and their tendency to accumulate in biologic as well as nonbiologic media, the organochlorine insecticides were replaced by the organophosphate insecticides for many uses in the early 1950s (Klaassen, Amdur, and Doull 1986). DDT is one of the best known, cheapest, and one of the most effective of the synthetic insecticides. The general population has sustained exposure to DDT and its derivatives with storage of some quantity of this insecticide in fatty tissues as a result of its introduction into commerce in the mid-1940s. The USEPA has restricted the use of DDT in the United States because of the ecological effects, potential effects of chronic exposure and storage of low levels of DDT in humans, and the development of resistant strains of insects. However, it is still used elsewhere worldwide. Humans may be exposed to organochlorine insecticides in contaminated air, soils, water, food, and breastmilk by inhalation, dermal contact, and ingestion exposure routes.

The persistence of organochlorines in the environment varies with each individual compound. Organochlorines, including aldrin, chlordane, endosulfan, and heptachlor, are reported as ranging from moderately persistent, with effectiveness ranging from 1 to 18 months, to persistent, retaining toxicity for years, perhaps as many as 50 to 100 years (Briggs and Council 1992). Lindane, DDT, DDE, DDD, dieldrin, endrin, and methoxychlor are persistent insecticides (Briggs and Council 1992).

Physical and chemical properties of DDT

Property	Value
Molecular weight	354.5 g/mol
Water solubility	0.0031 mg/L at 25 °C
Vapor pressure	8.3×10^{-6} mm Hg at 20 °C
K_{oc}	113,000 to 350,000
log K_{ow}	6.91
Henry's Law Constant	3.8×10^{-3} atm-m^3/mol at 20 °C

Toxicity

In general, the organochlorine insecticides are considered to be less acutely toxic, but have greater potential for chronic toxicity, than the organophosphate

and organocarbamate insecticides. The oral LD50 in male rats for a number of organochlorines ranges from 18 mg/kg for endrin to 5,000 to 7,000 mg/kg for methoxychlor, while the dermal LD50 ranges from 18 mg/kg for endrin to 2,510 mg/kg for DDT (Klaassen, Amdur, and Doull 1986). Acute hazard potential may be ranked approximately from highest to lowest as endrin, aldrin, dieldrin, chlordane, toxaphene, kepone, heptachlor, DDT, and methoxychlor Hazardous Substances Database (HSDB 1997).

Organochlorine insecticides are classified as neurotoxins; however, their mechanism of action is not the same as that of the organophosphates and organocarbamates. DDT is believed to act on the sensory and motor nerve fibers and the motor cortex, inducing repetitive firing in the presynaptic nerve membrane (Klaassen, Amdur, and Doull 1986). Signs and symptoms of acute DDT poisoning include paresthesia of the tongue, lips, and face, apprehension, hypersusceptibility to stimuli, irritability, dizziness, disturbed equilibrium, tremor, and tonic and clonic convulsions. Although the central nervous system (CNS) is the primary site of toxic action, primary pathologic changes resulting from subacute or chronic feeding are observed in the liver. Large doses of DDT in animal studies result in centrolobular necrosis of the liver, while smaller doses result in liver enlargement. Histologic changes in the livers of male rats fed diets containing 5 to 15 mg/kg or more for 6 months include hypertrophy, inclusion bodies, and cytoplasmic granulation (Klaassen, Amdur, and Doull 1986). DDT and related compounds induce mixed-function oxidase enzymes of the liver in several species, including humans and increases the incidence of liver tumors in rodent diet studies (Klaassen, Amdur, and Doull 1986).

Methoxychlor and lindane have low CNS toxicity. However, there have been a number of fatalities resulting from acute poisoning by the cyclodiene insecticides, considered CNS stimulants. The precise site and mechanism of toxic action for these compounds are unknown. Acute, subacute, and chronic toxicity studies of aldrin and dieldrin in experimental animals have shown the critical effects to be increased liver/body weight ratios and histologic changes in the liver, occurring at 0.5 mg/kg of dieldrin and 2 to 2.5 mg/kg of aldrin in rats (Klaassen, Amdur, and Doull 1986). Like DDT, all of the chlorinated cyclodiene insecticides are capable of inducing hepatic microsomal drug-biotransformation enzymes. Lindane and alpha-BHC are convulsants, while beta and delta-BHC are CNS depressants. The mechanism of neurotoxic action of these compounds has not been demonstrated.

There is a growing body of evidence which suggests that environmental chemicals, including many of the organochlorine insecticides, can disrupt the endocrine system by exhibiting estrogenic function, causing a cascade of biological effects. Endocrine disrupters interfere with the role of natural hormones in the body. Organochlorine insecticides considered to be estrogenic include DDT, DDE, kepone, heptachlor, chlordane, dieldrin, lindane, and toxaphene. Observed effects in animal studies have included disruption of female and male reproductive functions, including disruption of normal sexual differentiation, ovarian function, sperm production, and pregnancy as well as effects on the thyroid gland (USEPA 1997). Rats given DDT exhibited estrogenic effects. A contaminant of DDT (o,p'-DDT) was shown to compete

with estradiol for binding the estrogen receptors in rat uterine cytosol and estrogen receptors in mammary tumors (Klaassen, Amdur, and Doull 1986).

Depressed sperm counts may accompany excessive absorption of organochlorines. Aldrin and dieldrin have been reported to produce various effects on reproduction in a variety of species, such as decreased fertility and decreased viability of the young, thought to be related to hormonal imbalance (Klaassen, Amdur, and Doull 1986).

IARC has listed some of these agents as "probably carcinogenic to humans"(B2), although it also categorizes them as being inadequately assessed for human carcinogenic potential. Their carcinogenicity has been demonstrated in animal studies, but insufficient data exist from human studies. Organochlorine compounds are categorized by carcinogenicity below (IRIS 1997).

Carcinogenic Organochlorines	Noncarcinogenic Organochlorines
Aldrin	delta-BHC
alpha-BHC	Endosulfan I
beta-BHC	Endosulfan II
Lindane	Endosulfan Sulfate
Chlordane	Endrin
DDT, DDE, DDD	Methoxychlor
Dieldrin	
Heptachlor	

Toxicokinetics

Organochlorines are absorbed by the respiratory tract, skin, and gastrointestinal tract. The limited dermal absorption of DDT is significantly enhanced when dissolved in oils, fats, or lipid solvents. DDT and a major metabolic product, DDE, tend to accumulate in adipose tissue, eventually reaching equilibrium at a constant rate of intake. There is a close correspondence between lipid content of organs and concentration of DDT, DDE, and dieldrin in blood, kidney, liver, and adipose tissue (Klaassen, Amdur, and Doull 1986). Storage in fat is a type of detoxification, because it decreases the amount of chemical at the site of toxic action, the CNS. Following absorption in mammals, the metabolism of DDT includes dehydrochlorination to unsaturated DDE and substitution of hydrogen for chlorine yielding DDD (Klaassen, Amdur, and Doull 1986). Following exposures, DDT is slowly eliminated from the body at a rate of approximately 1 percent of stored DDT excreted per day (Klaassen, Amdur, and Doull 1986). DDT metabolites (DDD and DDE) are excreted primarily in urine and feces, and in breastmilk.

The more rapid metabolism of methoxychlor is achieved by O-demethylation and subsequent conjugation and excretion, catalyzed by microsomal enzymes in mammals (Klaassen, Amdur, and Doull 1986). Aldrin and heptachlor are metabolized by microsomal enzymes to their corresponding epoxides, and they can be equally or more toxic than the corresponding parent compound (Klaassen, Amdur, and Doull 1986). Therefore, the epoxide formation might be considered an activation reaction. These epoxides are stored in the adipose tissues of

humans and other animals. The epoxides may be further biotransformed to dihydrols, which can be conjugated and excreted in the (Klaassen, Amdur, and Doull 1986). Biliary and fecal excretion of the cyclodiene insecticides also occur. Lindane has been metabolized in rats by progressive dehydrochlorination, glutathione conjugation, and aromatic hydroxylation. Primary excretion of the metabolites occurs in the urine (Klaassen, Amdur, and Doull 1986).

Ecological effects

As a result of the bioconcentration of organochlorine insecticides in ecosystems, organisms at the top of natural food chains may sustain injury due to the gradual accumulations of residues in organisms that make up their food sources. Reproductive success of certain species of wild birds is adversely affected by exposure to DDT or its metabolites (Klaassen, Amdur, and Doull 1986). Eggshell thinning has been demonstrated following ingestion of DDT and related chlorinated hydrocarbon insecticides. In addition, the ability of DDT to enhance the metabolism of estrogen may impact reproductive success in birds by creating an endocrine imbalance affecting egg laying and nesting cycles (Klaassen, Amdur, and Doull 1986). Fish and some lower aquatic organisms are extremely sensitive to the acute toxicity of DDT.

Significant evidence of endocrine disruption exists for the following groups of organisms: snails, oysters, fish, alligators and other reptiles, and birds, such as gulls and eagles (USEPA 1997). Significant population declines as a result of exposure to endocrine-disrupting chemicals have been reported for alligators in Central Florida and some populations of marine invertebrate species.

References

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Phenanthrene
Cas No. 85-01-8

Potential sources and exposures

Phenanthrene is a polycyclic aromatic hydrocarbon (PAH). The reader should refer to the general profile on PAHs for exposure information.

Physical and chemical properties

Property	Value
Molecular weight	178.2 g/mol
Water solubility	1.00 mg/L at 21 ºC
Vapor pressure	6.8×10^{-4} atm at 25 ºC
K_{oc}	14,000 mL/g
log K_{ow}	4.46
Henry's Law Constant	1.59×10^{-4} atm-m^3/mol at 25 °C

Toxicity

There are no data on the toxicity of phenanthrene to humans (IARC 1983). Phenanthrene has been tested for carcinogenicity in laboratory animals by the oral, dermal, and subcutaneous routes of administration (as cited in IARC 1983); however, IARC (1983) and USEPA (IRIS 1992) concluded that data from available studies were inadequate to permit an evaluation of its carcinogenicity of phenanthrene. In addition, the results of short-term mutagenicity tests are equivocal. Nonetheless, current theories regarding the mechanisms of metabolic activation of PAHs predict that phenanthrene may have carcinogenic potential (IRIS 1992).

Toxicokinetics

In general, many polycyclic aromatic hydrocarbon can produce toxicity after inhalation, oral, or dermal exposure. Thus, it is believed that PAHs are absorbed after exposure by these routes. Because of their high lipid solubility, PAHs are believed to be distributed throughout the body. Relative to other tissues, they tend to localize in body fat and fatty tissues. PAHs are generally metabolized by the microsomal mixed function oxidase system and eliminated via the hepatobiliary tract.

Several metabolites of phenanthrene have been identified. They include the 1,2- 3,4- and 9,10-dihydrodiols, and the 1,2-diol-3,4-epoxide. The dihydrodiols displayed little or no tumor-initiating activity on mouse skin (IARC 1983). The epoxide was found to be mutagenic in bacterial and mammalian cells (IARC 1983). USEPA (1982) reported significant tumorigenic activity with the expoxide but not with phenanthrene itself in newborn mice.

Ecological effects

The reader is requested to review the toxicity profile for PAHs for information regarding ecological effects. Acute toxicity of phenanthrene to fish has been reported at levels of 4,500 mg/L and would probably be lower for sensitive species or for chronic effects.

References

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Polychlorinated Biphenyls (PCBs, Aroclors) Cas No. 1336-36-3

Potential exposure

Polychlorinated biphenyls (PCBs) are a family of man-made chemicals that contain 209 individual compounds (referred to as congeners). Some commercial PCB mixtures are referred to by their industrial trade name, Aroclor. Due to their thermal stability, inflammability, and dielectric capability, PCBs were used in electrical capacitors and transformers. Although PCBs are no longer manufactured or used in this country, they have entered the environment from accidental spills, leaks from transformers or capacitors, or mismanaged electrical equipment wastes. Due to their chemical properties, PCBs are persistent in the environment; they do not readily break down and are bioconcentrated in the food chain. Humans might be exposed to PCBs in contaminated air, water, soils or food, such as fish.

Physical and chemical properties

Property	Value
For Aroclor 1260	
Molecular weight	375.7 g/mol
Water solubility	0.0027 mg/L
Vapor pressure	4.05×10^{-5} mm Hg at 25 °C
K_{oc}	no data available
log K_{ow}	6.8
Henry's Law Constant	4.6×10^{-3} atm-m^3/mol at 25 °C

Toxicity

Exposure to PCBs has caused dermatologic effects, in particular, chloracne. Cases of severe chloracne were reported in by workers exposed for 2 to 4 years in which PCB air levels were between 5.2 and 6.8 mg/m^3 (ATSDR 1991). Other effects might include dry sore throat, skin rash, gastrointestinal disturbances, eye irritation, and headache with inhalation exposures below 0.15 mg/m^3 PCB. There is some evidence of liver cancer in humans when exposed to PCBs via the inhalation, gastrointestinal, or dermal pathways. Confounding factors in these studies include the simultaneous exposure to polychlorinated dibenzofurans. Higher blood PCB levels are associated with elevated serum triglyceride and/or cholesterol levels, as well as elevated blood pressure. Some of these effects are

reversible after termination of exposure, but the concentration of stored PCB in adipose tissue will dictate the rapidity with which this will take place.

There is evidence in both animals and humans that PCBs might be fetotoxic, resulting in decreases in birth weight, head circumference, and gestational age of the newborn. In addition, behavioral deficiencies have been observed in newborns exposed to PCBs in breastmilk.

Most genotoxicity and mutagenicity assays of PCBs have been negative. The carcinogenic effects of PCBs have been studied in rats and mice. The USEPA carcinogenic slope factor is based upon a data obtained from a chronic feeding study of PCBs to rats in which trabecular carcinomas and adenocarcinomas were observed. Based on the positive evidence for carcinogenicity of Aroclors 1254, 1260, Kanclor 500 and Clophen A-30 and A-60 in animals, along with adequate evidence in humans, the USEPA has categorized these PCBs as B2, or probable human carcinogens (IRIS 1993).

Toxicokinetics

Following oral exposure to PCBs, gastrointestinal absorption of these compounds is efficient, estimated to be close to 100 percent. Absorption via dermal and inhalation routes is not as efficient. The PCBs are poorly metabolized to more polar compounds, contributing to their long biological half lives. Distribution of PCBs follows a biphasic pattern: initially to muscle and liver, followed by redistribution to organs with high fat content, such as fat and skin. Excretion occurs primarily in the feces.

Ecological effects

Due to the former extensive use and stability of the PCBs, there is widespread occurrence of these compounds in soils and water. In general, the higher the degree of chlorination, the more resistant to biodegradation and the more persistent in the environment are the PCBs. Bioconcentration factors in aquatic species range from 26,000 to 60,000. Analyses of whole fish samples collected nationwide revealed PCB residues in 94 percent of all fish surveyed, at a mean concentration of 0.53 ppm.

It is well documented that PCBs interfere with reproduction in wildlife and in experimental animals.

References

Agency for Toxic Substances and Disease Registry. (1991). "Toxicological profile for selected PCBs," U.S. Department of Health and Human Services, Washington, DC.

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Polycyclic Aromatic Hydrocarbons (PAHs, polynuclear hydrocarbons)

Since the PAHs are rarely found individually in the environment and the effects on the environment and human health are not well defined for discrete PAHs, the reader is asked to refer to this toxicity profile for general information on the PAHs and to use the individual toxicity profiles for specific compounds.

Potential sources and exposure

The PAHs are a group of compounds that are formed during the incomplete combustion of coal, oil, gas, wood, and other organic compounds. Natural sources of PAHs include forest fires and volcanic eruptions. PAHs are ubiquitous in soil and are rarely found as individual compounds. The greatest exposure sources of PAHs to humans are active or passive inhalation of the compounds in tobacco smoke, wood smoke, and contaminated air. Exposure may also occur through ingestion of grilled or smoked foods, contaminated water or foods and through skin contact with soot, tars, or contaminated sediments.

Physical and chemical properties

The PAHs have been categorized by the number of aromatic rings in their chemical structure as well as by their carcinogenicity in laboratory animals. Although naphthalene is a two-ringed structure, it is frequently categorized as a PAH. The other compounds are listed below and are three-, four-, or five-ringed structures. PAHs commonly found in the environment are solids at room temperature and are virtually insoluble in water.

2-Ringed PAH	3-Ringed PAHs	4-Ringed PAHs	> 4-Ringed PAHs
Naphthalene	Acenaphthene	Benzo(a)anthracene	Benzo(b)fluoranthene
	Acenaphthylene	Benzo(a)pyrene	Benzo(k)fluoranthene
	Anthracene	Chrysene	Benzo(ghi)perylene
	Fluorene	Fluoranthene	Dibenz(a,h)anthracene
	Phenanthrene	Pyrene	Indeno(1,2,3-cd)pyrene

Toxicity

Within the large class of PAHs, there have been many structure-activity relationship studies to relate chemical structure to carcinogenic activity. Each of the environmentally relevant PAHs have been tested for their carcinogenicity in animal studies and the compounds are categorized by carcinogenicity in the following tabulation.

Carcinogenic PAHs	Noncarcinogenic PAHs
Benzo(a)anthracene	Acenaphthene
Benzo(a)pyrene	Acenaphthylene
Benzo(b)fluoranthene	Anthracene
Benzo(k)fluoranthene	Fluorene
Dibenz(a,h)anthracene	Fluoranthene
Chrysene	Naphthalene
Indeno(1,2,3-c,d)pyrene	Pyrene
	Phenanthrene

There are minimal data, animal or human, on the systemic toxicities of the PAHs and virtually no data on the acute effects of the compounds. Toxic effects that have been observed include a variety of skin lesions and noncancer lung diseases.

Toxicokinetics

Animal data indicate that the PAHs are readily absorbed after exposure by inhalation or oral intake and distributes to many tissues in the body. However, intestinal absorption of the PAHs is dependent upon the presence of bile in the stomach. The PAHs are absorbed via dermal exposure as shown by both human and animal studies, although very little is distributed to tissues (USEPA 1982). Following absorption, metabolism via the cytochrome P-450 monooxygenase system is required for detoxification to more water-soluble forms of the compounds for efficient elimination from the body. The unmetabolized PAHs are not believed to be carcinogenic. During the detoxification process, some PAHs are metabolically activated to their carcinogenic intermediates. These intermediates can then bind to cellular macromolecules such as DNA, RNA, and proteins, resulting ultimately in the induction of cancer. For any of the PAHs, however, the majority of the metabolism results in detoxified metabolites that are rapidly excreted.

Ecological effects

The PAHs as a group of contaminants constitutes the largest number of chemicals of interest identified at manufactured gas plant sites.

No standard freshwater toxicity tests have been reported for polycyclic aromatic hydrocarbons (except naphthalene) as a class or specific compounds. There are some data for bioconcentration during tests with model ecosystems, or for short exposure periods.

Lu et al. (1987) conducted studies with benzo(a)pyrene in a terrestrial-aquatic model ecosystem and observed bioconcentration factors after 3 days ranging from 930 for the mosquitofish to 134,248 for Daphnia pulex. Bioconcentration factors for Daphnia magna and Hexagenia sp. for a shorter time were 200 to 3,500. English sole and white suckers from populations with high frequencies of neoplasia had elevated levels of PAHs in their stomach contents.

Some PAH metabolites are carcinogenic, mutagenic, or teratogenic to organisms. Rather than enhancing detoxification, metabolism of some carcinogenic PAHs in induced animals could result in a higher steady-state level of toxic products (Steggeman 1981). Although studies with various carcinogens have demonstrated that chemicals can cause cancer in aquatic species, most attempts to demonstrate carcinogenesis by PAHs in aquatic species have produced equivocal results (Pliss and Khudoley 1975). Although recently there has been some evidence that PAHs can cause cancer in aquatic animals, there is to date no direct evidence of a single specific PAH induction of carcinogenesis in aquatic species (Neff 1979 and Steggeman 1981).

Studies in the Duwamish River, Boston Harbor, and Hudson River have identified populations of Dover sole and Atlantic tomcod with very high incidences of hepatocellular carcinoma (Varanasi 1989), and higher incidences of similar diseases have been reported for other environments. Although the etiology of such diseases in fish is uncertain, there is reason to suspect that the chemical environment is responsible, and PAHs have not been exonerated (Steggeman 1981). Bottom sediments in the areas that these fish populations inhabited contained elevated levels of PAHs.

The impacts of concern in the terrestrial environment include both direct toxicity and food-chain impacts. The toxic effects of PAHs in mammals can be inferred from the extensive toxicity testing work performed on laboratory animals. As with humans, the basic conclusion is that exposure to PAHs are only slightly to moderately toxic by acute exposure, but longer exposures to certain PAHs can result in cancer. Biomagnification in animal food chains is unlikely, however, since PAHs are readily metabolized.

References

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Pyrene
(Benzo(def)phenanthrene)
Cas No. 129-00-0

Potential sources and exposure

Pyrene is a PAH. The reader should refer to the general profile on PAHs for exposure information.

Physical and chemical properties

Property	Value
Molecular weight	202.2 g/mol
Vapor pressure	2.5×10^{-6} at 25 °C
Water solubility	0.135 mg/L at 25 °C
K_{oc}	38,000 mL/g
log K_{ow}	4.88
Henry's Law Constant	5.1×10^{-6} atm-m^3/mol at 25 °C

Toxicity

Pyrene is considered to be a skin irritant in humans (as cited in IRIS 1992). Pyrene has produced negative results in most mutagenicity assays (USEPA 1982). IARC (1983) concluded that there is limited evidence that pyrene is active in short-term mutagenicity assays. Pyrene is classified as a Group D carcinogen by the USEPA based on the lack of human carcinogenicity data and inadequate data from animal bioassays.

The RfD for oral exposure to pyrene is 0.03 mg/kg-day, based on the observation of kidney toxicity in mice that received subchronic dosing with pyrene by gavage (USEPA 1989 as cited in IRIS 1992). Confidence in the database is low due to the lack of supporting evidence from other subchronic, chronic, or developmental/reproductive studies.

Toxicokinetics

Human exposure to pyrene is almost exclusively through ingestion and inhalation although it can be absorbed through the skin. There are no pharmacokinetic data for pyrene in humans (USEPA 1980). Because of their high lipid solubility, PAHs are believed to be distributed throughout the body. Relative to other tissues, they tend to localize in body fat and fatty tissues.

Pyrene, like other PAHs, is apparently metabolized via the microsomal mixed function oxidase system in mammals.

Elimination of pyrene from rats exposed to a pyrene aerosol (500 mg/L, 0.3 to 0.5 mm particles) for 60 min was reported (Mitchell and Tu 1979 as cited in USEPA 1982) to rapidly occur primarily via the liver and biliary system. When 50 ug of pyrene was administered in a gelatin-saline suspension to two rats by stomach tube, approximately one-half of the administered pyrene was still present in the gastrointestinal tract after 24 hr (Mitchell and Tu 1979 as cited in USEPA 1982).

Ecological effects

The reader is requested to review the toxicity profile for PAHs for information regarding ecological effects. A no effect level of 5 mg/L was observed for trout in an acute (24 hr) exposure. Adequate data for characterization of toxicity to domestic animals and wildlife are not available.

References

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Silver

General background information

Silver is used in photographic materials, batteries, paints, and jewelry. Silver is used medically in dental amalgam and in medical supplies for burn treatment. Photographic materials are the major source of silver that is released into the environment. Trace amounts of silver are found in water from natural sources and industrial waste.

Pharmacokinetics

Studies in humans and animals indicate that silver compounds are absorbed readily by the inhalation and oral routes. Individuals and individual organs absorb silver selectively. The greatest concentrations are found in the reticuloendothelial organs. Silver undergoes oxidation and reduction reactions within the body and is excreted primarily via the fecal route (ATSDR 1990).

Human toxicological profile

Blue-gray discoloration of the skin has been observed in many individuals who have ingested metallic silver and silver compounds over periods of months to years. This condition is termed argyria. The pigmentation of the skin is primarily in sun-exposed areas. Silver-containing granules are also observed in the dermis. Gradual accumulation of 1 to 5 grams of silver will lead to generalized argyria. The discoloration is not known to be diagnostic of any other toxic effect (ATSDR 1990). Occupational exposure to silver dusts can lead to respiratory and gastrointestinal irritation. The average air level was estimated to range from 0.039 to 0.378 mg/m3. Duration of employment ranged from less than 1 year to greater than 10 years. Symptoms included abdominal pain, sneezing, stuffiness, and sore throat. Granular deposits were also observed in the conjunctiva and corneas of the eyes (Rosenman, Moss, and Kon 1979; Rosenman, Seixas, and Jacobs 1987). Medical case histories indicate that dermal exposure to silver and silver compounds for extended periods of time can lead to local skin discoloration similar in nature to the generalized pigmentation seen after repeated oral exposure. The amount of silver and the duration of exposure necessary to produce this effect have not been established (McMahan and Bergfeld 1983).

Mammalian toxicological profile

Oral doses of 1,680 mg/kg silver colloid resulted in the deaths of rats after 4 days (Dequidt, Vasseur, and Gomez-Potentier (1974). Ingestion of silver nitrate and silver chloride will also cause deposition and silver granules in the skin of animals (Walker 1971). Granules were observed in the eyes of rats exposed to silver nitrate in drinking water at doses of 222 mg/kg/day over 37 weeks. These doses also cause general deposition in other tissues (Matuk,

Ghosh, and McCulloch 1981). Mice given oral doses of 18.1 mg/kg/day silver nitrate for 125 days were observed to have silver deposits in their nervous systems. These animals were less active than unexposed controls (Rungby and Danscher 1984). Silver has been found in the brains of neonatal rats whose mothers received silver lactate on days 18 and 19 of gestation (Rungby and Danscher 1984). No studies were located that examine the reproductive effects of silver in animals or humans.

Genotoxicity

Silver is not mutagenic in bacteria but it has been found to cause DNA damage in mammalian cell culture (Robinson, Cantoni, and Costa 1982). No studies were located regarding cancer in humans or animals following oral, inhalation or dermal exposure to silver or silver compounds (ATSDR 1990).

References

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Zinc
Cas No. 7440-66-6

Potential sources and exposure

Zinc occurs in nature in the 0 and +2 valence states, although it is also found in four other stable valences. Metallic zinc is insoluble in water, although some zinc salts are soluble and found naturally in drinking water. Exposure to zinc in very low concentrations occurs daily through the diet. Average zinc intake through the diet ranges from 7 to 16.3 mg/day. Zinc is an essential trace element. Zinc is used in the manufacture of galvanized iron, bronze, white paint, rubber, glazes, enamel, glass, paper, and as a wood preservative. Exposure to zinc at higher levels can occur from drinking water or other liquids stored in galvanized metal containers.

Physical and chemical properties

Property	Value
Molecular weight	65.4 mg/l

Toxicity

Ingestion of excessive amounts of zinc above the recommended daily allowance for zinc of 15 mg may cause fever and gastrointestinal distress. Following acute, intermediate, or chronic ingestion of zinc, the primary effects in humans are pancreatic abnormalities, and gastrointestinal irritation. Ingestion of zinc has resulted in the reduction of HDL-cholesterol levels in humans. Oral exposure has been reported to impair immune and inflammatory responses. Anemia may occur after high-level acute, intermediate, or chronic oral exposure to zinc.

Inhalation exposure to zinc dust or fumes has been associated with pulmonary fibrosis and metal fumer fever. Acute high-level exposure to zinc oxide causes metal fume fever. Zinc oxide penetrates the alveoli, damages the lung tissue, and transiently impairs respiratory function. Metal fume fever is believed to be the result of an immune reaction to inhaled oxide particles. Chronic exposure to zinc has produced anemia. Zinc needs to be present at certain levels to predict fetal/developmental abnormalities or effects.

There is no evidence to indicate zinc and its compounds are associated with carcinogenicity in humans (IRIS 1992).

Toxicokinetics

It appears that zinc is absorbed via ingestion and inhalation. Zinc is widely distributed throughout the body and is found in high concentrations in male

reproductive organs, pancreatic islets, muscle, kidney, liver, and bone. Excretion of zinc is mainly though the gastrointestinal tract, though some of the zinc is reabsorbed. It is also excreted via urine, feces, sweat, hair, and saliva. Placental transfer of zinc may also occur. The half-life of zinc in humans in 200 to 400 days.

Ecological effects

Zinc is an essential micronutrient for all living organisms. Because zinc is essential, zinc is bioaccumulated by all organisms. The toxicity of zinc is dependent upon its chemical form and degree of interconversion among the various forms. Zinc will not be sorbed or bound unless it is dissolved, but bound zinc will dissolve in the digestive tract following the ingestion of particulates. The toxicity of undissolved zinc to a particular species depends on the feeding habits. Aquatic plants and most fish are relatively unaffected by suspended zinc in the water column. Both terrestrial and aquatic invertebrates and filter feeder fish might be adversely affected by ingestion of sufficient quantities of particulates containing zinc. The acute toxicity of zinc to aquatic animals is influenced by several parameters including increasing hardness, abundant dissolved oxygen, and low temperatures which lower the potential toxicity of zinc.

Reported acute toxicity testing for freshwater organisms indicates that insects are most resistant whereas cladocerans and the striped bass are the most sensitive to zinc. The reported mean genus acute value for cladoceran is 50.56 ug/l at a hardness of 50 mg/l. The final acute value representing zinc toxicity to freshwater species is 108.4 ug/l at a hardness of 50 mg/l.

The range of species mean acute values for saltwater invertebrates extends from 166 ug/l for embryos of the quahog clams, Mercenaria mercenaria, to 320,400 ug/l for adults of the clam, Macoma balthica. In general, early life stages of saltwater invertebrates and fish are more sensitive to zinc than juveniles and adults. The saltwater final acute value for zinc is 174.5 ug/l which is higher than the acute value of 166 ug/l for the quahog clam. Chronic toxicity values range from 47 to 852 ug/l and appear to be relatively unaffected by hardness.

Zinc was found to accumulate in freshwater animal tissues from 51 to 1,130 times the concentration present in the water (USEPA 1980). Steady-state zinc bioconcentration factors for 12 aquatic species range from approximately 4 to 24,000 (USEPA 1980).

Zinc bioconcentration from soil by terrestrial plants, invertebrates, and mammals, in values of 0.4, 8 and 0.6, have been reported. It has also been reported that phytotoxic tissue zinc levels ranging from 200 to 400 ppm. Studies have reported that 60 to 81 ppm of zinc in wheat and corn tissue is phytotoxic.

The tolerance of domestic livestock to zinc in animal feed ranges from 300 to 1,000 ppm (National Academy of Science (NAS) 1980). Zinc poisoning has occurred in cattle. In one outbreak, poisoning was caused by food accidentally contaminated with zinc at a concentration of 20 g/kg. An estimated intake of

140 g of zinc per cow per day for about 2 days was reported. The exposed cows exhibited served enteritis, and some died or had to be slaughtered. Some researchers have speculated that exposure to excessive amounts of zinc may constitute a hazard to horses. Findings in foals living near lead-zinc smelters suggest that excessive exposure to zinc may produce bone changes, joint afflictions, and lameness. In swine given dietary zinc at concentrations greater than 1,000 mg/kg, decreased food intake and weight gain were observed. At dietary levels greater than 2,000 mg/kg, deaths occurred as soon as 2 weeks after exposure. Severe gastrointestinal changes and brain damage, both of which were accompanied by hemorrhages, were observed, as well as changes in the joints.

References

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National Academy of Sciences (NAS). (1980.) *Drinking water and health.* Vol 3. Safe Drinking Water Committee, National Research Council. National Academy Press, Washington, DC.

United States Environmental Protection Agency. (1980). Environmental Criteria and Assessment Office, "Ambient water quality criteria for zinc," NTIS PB81-117897, Cincinnati, OH.

Appendix E
Human Exposure Equations

This subsection calculates a separate dose of each contaminant for a receptor based on:

Exposure Point Concentrations (EPC)

The EPCs are the measured or modeled chemical concentrations for each pathway. The EPCs are unique to each scenario.

Exposure Assumptions

The *exposure assumptions* describe the receptor and the conditions under which the receptor contacts the exposure point concentrations. Unless otherwise indicated, these assumptions are standard U.S. Environmental Protection Agency (USEPA) defaults (USEPA 1989, USEPA 1992c).[1]

Averaging Time (USEPA 1989)

The *averaging time* is the time over which the receptor is exposed for noncarcinogenic risk and is a lifetime for carcinogenic risk. It will vary depending upon the assumptions used.

For most exposure routes, the following equations assume that the absorption of a COC into the bloodstream from the gastrointestinal tract and lungs is 100 percent. Therefore, these equations are not adjusted in any manner. However, the dermal route of exposure will result in an absorption of a COC into the bloodstream which is less than 100 percent. The equation describing the dose from this exposure route is appropriately adjusted (USEPA 1992c).

The following equations are used to estimate doses. The inhalation route is evaluated based on the concentration of contaminants in the air, not a dose (USEPA 1989).

[1] A complete list of references is located at the end of the main text.

Ingestion of Chemicals in Drinking Water

$$Intake(mg/kg-day) = \frac{EPC_W \times IR \times EF \times ED}{BW \times AT} \qquad \text{(E1)}$$

where

EPC_w = Chemical concentration in water (mg/liter)

IR = Ingestion rate (liters/day)

EF = Exposure frequency (days/year)

ED = Exposure duration (years)

BW = Body weight (kg)

AT = Averaging time (period over which exposure is averaged: days)

Incidental Ingestion of Chemicals in Surface Water While Swimming

$$Intake(mg/kg-day) = \frac{EPC_W \times CR \times ET \times EF \times ED}{BW \times AT} \qquad \text{(E2)}$$

where

EPC_w = Chemical concentration in water (mg/liter)

CR = Contact rate (liters/hour)

ET = Exposure time (hours/event)

EF = Exposure frequency (events/year)

ED = Exposure duration (years)

BW = Body weight (kg)

AT = Averaging time (period over which exposure is averaged: days)

Dermal Contact with Chemicals In Water

In order to convert an external dose to an absorbed dose in the dermal pathway, a dermal absorption factor is applied to the EPC. Dermal absorption factors are selected based on EPA Region 1 Guidance and simple structure activity relationships.

$$Absorbed\ Dose(mg/kg-day) = \frac{EPC_W \times CR \times ET \times EF \times ED}{BW \times AT} \qquad \text{(E3)}$$

where

CW	=	Chemical concentration in water (mg/liter)
SA	=	Skin surface area available for contact (cm^2)
PC	=	Chemical-specific dermal permeability constant (cm/hour)
ET	=	Exposure time (hours/day)
EF	=	Exposure frequency (days/year)
ED	=	Exposure duration (years)
CPC_f	=	Volumetric conversion factor for water (1liter/1,000cm^3)
BW	=	Body weight (kg)
AT	=	Averaging time (period over which exposure is averaged: days)

Ingestion of Chemicals in Soil (and Sediment)

$$Intake(mg/kg-day) = \frac{EPC_S \times IR \times CF \times FI \times EF \times ED}{BW \times AT} \quad \text{(E4)}$$

where

EPC_s = Chemical concentration in soil (mg/kg)

IR = Ingestion rate (mg soil/day)

CF = Conversion factor (10^{-6} kg/mg)

FI = Fraction ingested from contaminated source (unitless)

EF = Exposure frequency (days/year)

ED = Exposure duration (years)

BW = Body weight (kg)

AT = Averaging time (period over which exposure is averaged: days)

Dermal Contact with Chemicals in Soil

In order to convert an external dose to an absorbed dose in the dermal pathway, a dermal absorption factor is applied to the exposure point concentration. Dermal absorption factors are selected based on structure activity relationships.

$$Absorbed\ Dose(mg/kg-day) = \frac{EPCs \times CF \times SA \times AF \times ABS \times EF \times ED}{BW \times AT} \quad \text{(E5)}$$

where

CS	=	Chemical concentration in soil (mg/kg)
CF	=	Conversion factor (10^{-6} kg/mg)
SA	=	Skin surface area available for contact (cm^2/event)
AF	=	Soil to skin adherence factor (mg/cm^2)
ABS	=	Absorption factor (unitless)
EF	=	Exposure frequency (events/year)
ED	=	Exposure duration (years)
BW	=	Body weight (kg)
AT	=	Averaging time (period over which exposure is average: days)

Inhalation

Use measured or modeled air concentrations for comparison to USEPA reference calculations available in IRIS database at *www.epa.gov/ngispgm3/iris.* This is EPA's Integrated Risk Information System (see Appendix B).

Food Pathway Ingestion of Contaminated Fish and Shellfish

$$Intake(mg/kg-day) = \frac{CPC_f \times IR \times FI \times EF \times ED}{BW \times AT} \qquad \text{(E6)}$$

where

CPC_f = Contaminant concentration in fish (mg/kg)

IR = Ingestion rate (kg/meal)

FI = Fraction ingested from contaminated sediment (unitless)

EF = Exposure frequency (meals/year)

ED = Exposure duration (years)

BW = Body weight (kg)

AT = Averaging time (period over which exposure is averaged: days)

Appendix F
Hypothetical Example

Appendix F provides the examples used in the text in a continuous format. This is meant for illustrative purposes only and is provided here for the reader's convenience.

Description of the Dredged Material Management Activity

A local marina has proposed dredging 10 new slips. The existing water depths at the slips is 1.5 m (5 ft) mean lower low water (MLLW). Each slip will be 15 × 6 m (50 × 20 ft) and dredged to a depth of 3 m (10 ft) MLLW with a 0.6-m (2-ft) over-dredge allowance. The project will also require dredging of the channel resulting in an estimated 76,500 cu m (100,000 cu yd) of dredged material. A clamshell dredge will remove the material to a hopper barge for transport to an offshore unconfined management area for which a site designation report is available. The water depth near the site averages 30 m (100 ft), and there is low to moderate wave energy.

Description of the Habitat Surrounding the Management Site

The risk assessor used the following questions to guide the description of the habitat at and near the management area (disposal site) where the dredged material from the marina and channel will be transported.

a. What is the size of the management area (disposal site)?

b. What is the size of the local water body?

c. Are there fishery breeding, nursery, or feeding areas near the site?

d. Is the site near or adjacent to seasonal migration pathways for fish, mammals, or piscivorous birds?

e. Are there biological reefs near the site (shellfish reefs, coral reefs) or other particularly productive benthic environments?

f. Is the site near a wetland such as a salt marsh, Typha marsh, tidal flat, or flood plain?

g. Is the site near a productive commercial or recreational fishery?

h. Are there habitats identified by local, state, or Federal agencies for special protection such as critical habitat for endangered species, a national seashore park, or a state wetland refuge near the site?

i. Are there Federal, state, or endangered species near the site?

The management area for this dredging project is in a coastal bay that is approximately 8 × 3 km (5 × 2 miles) and connects to the open ocean through a broad mouth. The management site is 5 km (3 miles) offshore. The nearshore environment includes an extensive salt marsh. The bay has a sand and silt bottom and a stratified, seasonal thermocline. There is a winter flounder fishery near the site. There are migratory species, including winter flounder and mackerel, in the area. There are no endangered species found near the site.

Identification of Species and Humans that May Use Habitats

Table F-1 is a summary list of species identified at or near the potential dredged material management site. It characterizes the species by habitat (e.g., planktonic, benthic) and by function within the ecosystem. Most of this information will have been assembled during the site designation process.

Tabulations such as these allow the risk assessor to judge the diversity of habitats among the aquatic community and provide some sense of general diversity and ecological function at the management site. Note that the species in this table, while they occur at or near the site, will not necessarily be selected as receptors for further analysis. For example, at most sites it is unlikely that phytoplankton will receive more than a short-term exposure to the dredged materials (primarily during disposal), because most of the contaminants potentially associated with dredged materials have a high affinity for sediment particles and low solubility.

Table F1
Species List for Management Area and Adjacent Areas

Receptor	Common Name	Functional Group
Phytoplankton		Primary producer
Asterionella		Primary producer
Melosira		Primary producer
Nitzschia		
Epibenthic Animals		
Homerus americanus	Lobster	Scavenger/predator
Crassostrea virginica	Oyster	Filter feeder
Infauna/Benthic Animals		
Mya arenaria	Soft shell clam	Filter feeder
Mercenaria mercenaria	Hard shell clam	Filter feeder
Cardium edule	Cockle	Filter feeder
Gammarus duebeni	Amphipod	Deposit feeder
Nereis virens	Sandworm	Scavenger/predator
Fish		
Anguilla rostrata	Eel	Predatory fish
Scomber scombrus	Mackerel	Migratory pelagic feeder
Pseudoplueronectes americanus	Winter flounder	Bottom feeding fish

In addition to these species, there are also humans who use the area around the site, including workers involved in dredging, transport, or management of the material, fishermen, and boaters. Because there is a winter flounder fishery near the site, other individuals may be exposed through fish consumption.

Identifying Contaminants of Concern (COC)

For the marina project under consideration, five contaminants found in the dredging material intended for the offshore management site met the criteria for Tier I identification of COCs. Specifically, cadmium, lead, mercury, endosulfan, and PCBs are potential contaminants of concern because they are present in the material and have known toxicological effects.

The tabulation below provides the WQC and the predicted concentrations for the potential COCs from Tier II evaluations. The evaluation revealed that neither lead nor cadmium have WQC for the protection of humans from consumption of organisms. These two contaminants must, therefore, be retained as COCs.

The remaining contaminants, mercury, endosulfan, and PCBs, have all WQC including: acute criteria for the protection of aquatic life; chronic criteria for the protection of aquatic life; criteria for the protection of humans from organisms only; and criteria for protection of humans from water and organisms. Among these three potential contaminants, the predicted water concentration of total PCBs from the dredged material exceeded the criteria. Therefore, total PCBs were retained as a COC.

A theoretical bioaccumulation potential could not be calculated for mercury because it is an inorganic compound. Therefore, a Tier III evaluation was necessary to determine compliance. The Tier III evaluation revealed that bioaccumulation of mercury in the dredged material was less than that of the reference sediment, and it was screened out as a COC.

Because endosulfan is a nonpolar organic compound, a theoretical bioaccumulation potential (TBP) could be calculated, but the TBP, in this case, did not exceed that of the reference sediment. In addition, no synergism with other potential COCs was suspected, and endosulfan was screened out as a COC.

At the end of the three-tiered evaluation, three contaminants in the dredged material, cadmium, lead, and PCBs, were selected as contaminants of concern for the risk assessment. This continuous example will carry total PCBs through the risk assessment.

Contaminant	Saltwater Criterion Acute Conc. (ug/L)	Saltwater Criterion Chronic Conc. (ug/L)	Criteria for Human Water and Organisms	Protection of Health Organisms Only	Predicted Contaminant Concentration	COCs Retained
Cadmium	43	9.3	10	NA	10.4	X
Endosulfan	0.034	0.0087	74	159	0.0067	
Lead	220	8.5	50	NA	14.7	X
Mercury	2.1	0.025	0.146	0.14	0.019	
PCBs	10	0.03	7.90E-05	7.90E-05	1.2	X

NA = Not available
Reference: USEPA (1999). "National recommended water quality criteria,", EPA/822-Z-99-001, Office of Water, Washington, DC.

Description of Potential Release Mechanisms

During this dredged material management operation, there are several potential release mechanisms which could result in exposure to COCs. Once the material has reached the management area, sediment can become suspended in

the water during placement. The area is a low-to-moderate energy environment, has a seasonal thermocline (indicating little surface to bottom mixing during summer), and is generally depositional. There is some potential for resuspension of the sediments and advection through wave or storm action and during winter with the breakdown of the seasonal thermocline. There is also potential for diffusion from pore water and advection offsite. These mechanisms could bring the potential COCs into contact with receptors.

Description of Complete Exposure Pathways

The risk assessor used the following questions to guide the determination of complete exposure pathways between the proposed dredged material and the potential receptors:

a. Could contaminants reach receptors via direct contact?

b. Are one or more receptors inhabiting or using an area where contamination exists or will exist?

c. Is the location of contamination such that one or more receptors could contact it currently or in the future?

d. Are there advective or dispersive processes which may deliver the contaminant to a receptor or habitat?

e. Could contaminants reach receptors via indirect contact?

f. Is contamination bioaccumulative or bioconcentratable?

g. Are there higher-order predators which may accumulate the contaminant?

h. Could contaminants reach receptors or habitats via groundwater?

i. Can contaminants leach into groundwater?

j. Does groundwater discharge to aquatic habitats? Are contaminants present at surface sediments?

k. Can contaminants be leached or eroded from surface sediments or soil?

The answers to these questions indicate that there is a benthic community with potential for direct contact and ingestion of sediments by invertebrate organisms at the management area. There is then potential for bioaccumulation to higher-order predators through ingestion of the benthic organisms. There is some potential for bioconcentration of COCs from suspended sediments in the water column to forage fish and zooplankton, given the moderate vertical mixing which may occur at the site in winter. The management option does not have an effluent discharge, so there is minimal likelihood of dissolved contamination in the water column (there is a potential for exposure in the water column during disposal, but

it is of short duration). There is a commercial fishery, winter flounder, which results in a complete pathway to humans through ingestion of flounder. The management area is too far offshore (5 km (3 miles)) to consider groundwater discharge as a likely exposure pathway. Also, the management option does not result in sediment exposures at the water surface as might be the case for an offshore containment island.

Selecting Human and Ecological Receptors

Ecological receptors

The potential receptors in the management site include the invertebrate community that lives on or in the sediments (the benthos), fish species that inhabit the bay for part of their life cycle or as a foraging area, and the plankton community of invertebrates, fish larvae, and algae that are suspended in the water column and carried with the tidal currents into and out of the bay.

Based on the data available for the site, it is clear that the focus of the analysis should be on animals that have direct contact with the sediments. These animal communities (both invertebrate and fish) tend to reside longer in particular areas than do plankton (carried with the currents) or fish that inhabit the water column (e.g., blue fish). Specifically, the environmental receptors which are emphasized in this analysis are the benthic invertebrate community and the demersal (bottom) fish community.

Within the demersal fish community, this risk assessment uses the winter flounder (Pseudopleuronectes americanus) as the representative species because it is the most commonly occurring species in the area, supports a major commercial fishery in the bay, and is a major predator on bottom dwelling organisms.

Human receptors

The likely human receptors include consumers of winter flounder from the commercial and recreational fishery.

Characterization of Ecological Receptors - Winter Flounder

The winter flounder is a coastal demersal species with a primary range in cold-temperate boreal waters. Winter flounder occur at depths from the intertidal to 150 m and on hard or soft mud, clay, sand, or pebble bottoms of bays, estuaries, and coastal waters (Bigelow and Schroeder 1953).[1] Perlmutter (1947) suggested the existence of many discrete local stocks based on several key

[1] References are listed following the main text.

observations: demersal eggs, nondispersive larvae, juvenile phases, and complete lack of adult mixing with other stocks.

Winter flounder spawn in most estuaries from Chesapeake Bay through the Gulf of Maine from midwinter to early spring (Azarovitz 1982). It is believed that winter flounder return to the same spawning location year after year National Marine Fisheries Service (NMFS) (1986). Winter flounder eggs are demersal and adhesive, and therefore the spawning and nursery areas for the species should coincide.

In areas north of Cape Cod, winter flounder remain in bays and harbors year-round, moving into deeper holes and channels during the warmest weather (Azarovitz 1982).

Winter flounder feed by sight near the bottom. For example, Pearcy (1962) showed that fish fed in a dark room did not eat until zooplankton died and sank to the bottom. Field observations confirmed that feeding occurs during the day. These organisms are clearly bottom dwellers who spend significant portions of their lives in close contact with sediments.

It is also significant that winter flounder eat bottom-dwelling organisms because the consumption of these organisms provides another potential exposure pathway. Several investigators (Pearcy 1962; MacPhee 1969; Frame 1972) noted that they are omnivorous, opportunistic feeders, and prey upon polychaete worms, amphipod and isopod crustaceans, pelecypods, and plant material.

Note that this example continues with assessing risk to winter flounder. The risk assessment should similarly address other selected receptors such as a representative benthic organism(e.g., softshell clams) or water-column organisms which may concentrate COCs from suspended sediments.

Evaluating the Assessment End Point, Health, and Maintenance of Local Flounder Populations

Consultation with the State Division of Marine Fisheries and the Save The Embayment Association (a citizen's action group) indicates that the area around the planned dredged material management site is a commercial flounder fishery. These groups are concerned that the disposal of dredged sediments from the marina slips may adversely affect flounder populations.

The assessment end point "health and maintenance of local flounder populations" is a reasonable assessment endpoint and it meets the evaluation criteria.

a. Ecological relevance. Flounder are major bottom feeders in this section of the Bay. Flounder populations generally play a major role in such marine ecosystem level properties as maintenance of invertebrate diversity and nutrient cycling.

b. Economic importance. Flounder are important economically in this portion of the bay. They constitute a commercial fishery year round and an important recreational fishery during summer in nearshore waters.

c. Measurable. The health and maintenance of local fish populations are measurable quantities.

d. Susceptible and sensitive to chemical induced stresses. There are toxicological and field studies supporting the sensitivity of fish to chemically induced stress.

e. Unambiguously defined. The health and maintenance of local fish populations is clearly distinct from assessment of migrating fish or wide ranging fish. The term "local" means populations whose feeding and migrating range is generally on the same scale as the area of the continental shelf proximate to the dredged material management site.

f. Logically and practically related to the management decision. Flounder live and feed near or on the sediments and are continuously exposed to surface water. Their protection as a local resource will be affected by management decisions regarding dredged material disposal in this region of the shelf.

Establishing an Appropriate and Relevant Measurement End Point

For PCBs, body burdens in flounder are a reasonable measurement end point. The flounder feed directly on benthic, sediment dwelling organisms that can bioaccumulate PCBs. Note that for other COCs this may not be a good end point. For example, the COCs, also include lead which does not biomagnify.

Attribute	Flounder Body Burdens of PCBs
Closeness of correspondence to the assessment end point	Moderate - the measurement of body burdens is not a direct measure of fish health or reproductive capacity
Site specificity	Strong - the fish probably acquire body burdens due to exposure to site-related contaminants
Correlation of stressor to response	Moderate - there is evidence in the literature indicating relationships between body burdens of COCs and changes in fish physiology, reproduction, and growth
Availability of an objective measure for judging environmental harm	Moderate - there are no promulgated standards for protection of ecological receptors based on body burdens. However, the USACE assembled a "residue effects" database for various contaminants
Sensitivity of the measurement end point for detecting changes	Moderate - the literature indicates a wide range in tolerance among fish species for body burdens of various COCs
Quantitative	Strong - the measurement is quantitative
Use of a standard method	Strong - there are accepted methods for analysis of COCs in tissue

Initial Estimate of Exposure Point Concentration for Total PCBs

The risk assessor has calculated the upper 95th-percent confidence limit on the arithmetic mean concentration of total PCBs based on Tier I measurements. This value is 1 ug total PCB/g sediment. The risk assessor has decided that the area of influence is equal to about one tidal excursion based on the description of the local environment as moderately energetic. The State Department of Marine Fisheries provided local oceanographic information to calculate the tidal excursion lengths. The management area and its area of influence are collectively referred to as the disposal site area.

Estimating a Body Burden in Winter Flounder

The dredged material management area and its area of influence (defined previously as the area within one tidal excursion of the site) is approximately equal to one-half the summer foraging area of the winter flounder, based on observations made by the state's Department of Marine Resources. This species is a selected receptor, based on its commercial importance.

The proposed site is within the State Statistical Fishery Area 4 and is 2 percent of that area.

As indicated earlier, the upper 95th-percent confidence limit of the arithmetic average total PCB concentration in the sediments from the proposed dredging project area is 1ug total PCB /g sediment.

The upper 95th-percent confidence limit of the arithmetic average of total PCB in sediments at the reference site is 0.10 ug total PCB/g. The assessment assumes that this is the exposure point concentration for winter flounder when foraging away from the site and its area of influence.

The average fraction lipid of a flounder is 0.1, based on hypothetical data provided by a fisheries agency.

Therefore, the average sediment exposure concentration of total PCB, Cs, at the disposal site is:

$$C_s = (1 \text{ H} 0.5) + (0.1 \text{ H} 0.5) = 0.55 \text{ ug total PCB/g sediment}$$

The state has also supplied data indicating that the fraction organic carbon in sediments in the area is 0.05 (5 percent).

A locally calculated biota sediment accumulation factor (BSAF) is 3, based on USEPA studies of PCB in flounder and sediment in this bay. The projected body burden (weight wet), Ca, to a flounder exposed to this total PCB concentration in sediments of 5 percent organic carbon is:

$$C_a = (0.55/\text{Foc}) \text{ H BSAF H (Fl)}$$
$$= (0.55/.05) \text{ H3 H0.1}$$
$$= 3.3 \text{ ug total PCB/g wet weight flounder tissue}$$

This body burden value can be used in both human health and ecological risk assessments.

This example could have used a different species such as lobster. In that case, the general method would remain the same, but parameters such as foraging area, bioaccumulation factor, and fraction lipid would differ. Also, the example is relatively simple in that it does not address differential uptake and storage of PCB congeners among tissues. In some instances, it may be important to estimate uptake in organs other than muscle. For example, lobster hepatopancreas has a different fraction lipid than lobster muscle. In a human health risk assessment, where some individuals in a population may consume the hepatopancreas, it becomes important to calculate a separate concentration for that tissue based on its particular lipid content.

Selection of a Toxicity Factor for Exposure of Winter Flounder to Total PCBs

Black et al. (1998) assessed the effects of PCBs on the reproduction of a fish using Fundulus heteroclitus (marine minnow) as an experimental organism. They measured a Lowest Observed Adverse Effect Level (LOAEL) at 3.8 ug PCB/g wet weight and a No Observed Adverse Effect Level (NOAEL) of 0.76 ug PCB/g wet weight. The risk assessor chose a body burden of 0.76 ug PCBs/g wet weight as the toxicity factor. This is an appropriate toxicity factor because:

a. It addresses toxicity to total PCBs, the COC.

b. It is from a study which includes the measurement of a NOAEL as well as a LOAEL.

Black et al. (1998) describe the end points in the study as female mortality and decreased egg production, therefore, the toxicity factor relates to the assessment end point "Health and Maintenance of the Local Flounder Population."

Risk to Flounder

The appropriate method to assess risk to flounder is to compare a measured effect level for body burden of PCBs in flounder to the calculated flounder body burden. As indicated earlier, the selected toxicity factor is 0.76 ug PCB/g wet weight. This is less than the 3.3 ug PCB /g body tissue concentration calculated for winter flounder in this example. Therefore, the assessment shows that there is potential for risk to the selected receptor, winter flounder. At this point, the risk assessor and risk mangers can:

a. Accept the initial conclusion and employ risk management activities.

b. Employ more complex fate and transport models and perhaps a more complex food chain model and recalculate risk.

The conclusion of risk from the initial estimates has various sources of uncertainty including:

a. Uncertainty concerning the actual foraging area of a flounder.

b. Uncertainty concerning the BSAF - the assessment used the recommended BSAF of 3 which may be overly conservative. A more sophisticated food chain model may give a more realistic estimate of body burden.

c. Uncertainty associated with possible interspecies differences between the experimental organism, *Fundulus heteroclitus*, and the flounder.

d. All the models used in the assessment are linear. Therefore, a simple sensitivity analysis can be performed using the ranges of various parameters.

Note that this estimate of potential risk applies to PCB exposures. The risk from the other COCs at this hypothetical site (PAHs and mercury) should be estimated as well. Also the risk characterization is iterative. At this point, the risk assessor may want to implement more sophisticated estimates of sediment concentrations using data intensive modeling. The assessor may also use a more sophisticated food chain model (e.g., Appendix C).

Description of Indirect Pathway - Consuming Winter Flounder

The management site is within a larger area representing a winter flounder commercial fishery. The site is close enough to shore to be a recreational fishery as well (although this example carries through only the commercial fishing scenario).

The flounder are landed at a medium sized city on the local bay, and the consumers are the people in the local metropolitan area. The State Department of Marine Fisheries indicates that little, if any, of the flounder are exported to a larger area.

Body Burdens in Winter Flounder

As indicated earlier, the risk assessor has identified a population in the area potentially exposed to PCBs from flounders in a commercial catch. The proposed disposal site will influence a fraction of this flounder catch. As described earlier, a tissue concentration of total PCBs can be calculated for flounder, based on measured sediment concentrations and observed biota-to-sediment concentration factors. These calculations resulted in a wet weight tissue concentration of 3.3 ug total PCB/g flounder tissue for flounders foraging over the disposal site. This is the EPC for total PCBs in the human health risk assessment.

Calculation of Fraction Ingested (FI) by Humans Based on Fishery Statistics for Consumption of Commercially Caught Flounder

The State Division of Marine Fisheries' winter flounder catch statistics indicate that 30 percent of all of the flounder landed in the State come from Statistical Area 4. For this example, Area 4 contains the hypothetical dredged material disposal site and its area of influence. It is known that the foraging area of a flounder is approximately 2 percent of Area 4.

Therefore

FI = 0.02 H0.3

FI = 0.006

In this case, the FI for the local metropolitan consumer of commercially harvested flounder is 0.006. Six-tenths percent of the flounder consumed by these receptors will be impacted by the dredge-management site. If there is reason to believe that the disposal site is preferentially attractive to flounder, this calculation will change accordingly.

Intake Calculation for the Consumption of Commercially Harvested Flounder

The risk assessor will calculate a potential average daily dose of total PCBs due to consumption of winter flounder exposed to the disposal site. The EPC (concentration of total PCBs in the flounder from the area of the site) and FI (fraction of the total catch from the area of the site) have been calculated previously. Note that the EPC is generally expressed as ug/g, although in the intake equation, it is necessary to convert that to mg/kg. The State Department of Marine Fisheries has indicated, in this hypothetical example, that a flounder ingestion rate of 0.11 kg per meal is a conservative estimate of flounder consumption.

$$ADD_{pot}\ (mg/kg/day) = \frac{EPC \times Abs \times IR \times FI \times EF \times ED}{BW_{avg} \times AT}$$

where:

EPC = (3.3 ug/g) = 3.3 mg/kg

Abs = 1

IR = 0.11 kg/meal

FI = 0.006

EF = 52 meals/year

ED = 9 years

BW_{avg} = 70 kg

AT = 70 years (365 days/year) = 25,550 days

$$ADD_{pot}(mg/kg/day) = \frac{3.3\ mg/kg \times 0.11\ kg/meal \times 0.006 \times 52\ meals/yr \times 9\ yrs}{70\ kg \times 25{,}550\ days}$$

$$ADD_{pot} = 5.6 \times 10^{-7}\ mg/kg/day$$

This is the incremental lifetime average daily intake for the consumption of commercially harvested flounder using conservative, reasonable maximum exposure assumptions.

Carcinogenic Risk Estimate for Consumption of Flounder

ILCR = Lifetime Average Daily Intake H CSF

Lifetime average daily intake	$= 5.6 \text{ H} 10^{-7}$ mg/kg/day
CSF for total PCB	$= 7.7$ (mg/kg/day)$^{-1}$
$ILCR_I$	$= 4.3 \text{ H} 10^{-6}$

The USEPA generally considers risks in the range of 10^{-6} to 10^{-4} as not indicating a potential human health risk. Therefore, exposure to total PCBs due to the proposed dredging project is unlikely to present a carcinogenic risk to the local human populations. However, this example calculates only risk from exposure to total PCBs. The summed ICLR due to exposure to PCBs and other COCs may present an unacceptable risk.

Note that there is uncertainty associated with this risk estimate because the USEPA currently emphasizes the need for congener specific analyses in assessing risk from PCB exposure.

Appendix G
Glossary of Terms

Assessment end points - valued characteristics of a management site or adjacent ecosystem that should be protected.

Average daily potential dose (ADD) - the dose of contaminants that enters the human body through the gastrointestinal tract following consumption of contaminated seafood.

Biota-sediment accumulation factor (BSAF) - expresses the accumulation of contaminants from sediments to the biota.

Body burden - the concentration of a contaminant of concern per unit body weight or per unit body lipid.

Carcinogenic slope factor (CSF) - expresses the carcinogenicity of a compound.

Complete exposure pathways - a physical, chemical, or biological mechanism, or some combination which may transport a contaminant from a source, such as sediment, to a specified human or other organism such as a commercial fish species or an endangered aquatic bird.

Conceptual model - an integration of existing information which attempts to identify the contaminants and their sources, describe the pathways by which they may reach humans or other organisms, and specify which humans or organisms might be linked to the contaminants by these pathways.

Dietary concentration - a contaminant of concern in the prey organism of a receptor.

Direct exposure pathways - dermal contact and ingestion of contaminated sediments or surface water.

Dose - the amount of a contaminant of concern ingested per unit body weight of the receptor per day.

Ecological exposure assessment - builds upon the qualitative descriptions in the conceptual model to calculate a quantitative estimate of the exposure of selected receptors to the contaminants of concern.

Exposure point concentrations - estimates of the concentrations of the contaminants of concern in environmental media to which the selected receptors may be exposed along the completed pathways.

Exposure scenarios - detailed descriptions of a human receptor's activities which result in exposure to the contaminants of concern; the pathway and route by which the human receptor contacts contaminants of concern; physical, chemical, and biological factors which affect the amount of the contaminant contacted or ingested.

Indirect exposure pathways - ingestion of seafood (finfish and shellfish, from marine or freshwater sources) which contains contaminants of concern.

LOAEL - the lowest concentration, dose, or body burden in a particular study for which adverse effects are reported.

Measurement end points - discrete observations that can be related to the assessment end point.

NOAEL - the highest concentration, dose, or body burden in a particular study for which no observable adverse effects are reported.

Problem formulation - a systematic planning stage that identifies the major factors considered in the assessment, and establishes its goals, breadth, and focus.

Receptors - humans or organisms that might be exposed to the contaminants via direct or indirect pathways.

Reference dose - the toxicity value used most often in evaluating noncarcinogenic effects, resulting from exposures to chemicals. Defined as an estimate of a daily exposure level for the human population, including sensitive subpopulations (such as elderly and children) that is likely to be without an appreciable risk of adverse effects during a lifetime.

Representative human receptors - humans who have a complete exposure pathway as described in the conceptual model, and whose exposure is likely to represent a reasonable worst case exposure to the COCs.

Representative ecological receptors - organisms whose life histories and habitat requirements fairly represent the range of habitats and life histories for those organisms with complete exposure pathways found near the dredged material management site.

Site characterization - a general description of the environmental setting.

Toxic end points - the type of effect (e.g., survival, growth, reproduction, etc.) associated with each toxicity factor.

Toxicity factor - environmental concentration, dose, body burden, or dietary concentration associated with a particular effect.

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13. ABSTRACT ***(Maximum 200 words)***

This document provides guidance for conducting ecological and human health risk assessments at aquatic sites potentially impacted by dredged material management activities.

Risk assessment is the process of evaluating the impact of a chemical or physical condition upon the health of individual humans or the environmental well-being of a population or community of animals and plants. The former is called human health risk assessment, and the latter ecological risk assessment.

The project manager should decide to apply a risk assessment within the context of the site selection process and/or the four-tiered evaluation of dredged material, or when there are unresolved issues with regard to potential human or ecological exposures. It is most applicable to projects which have:

a. Reached Tier IV and concern about specific bioaccumulative compounds or toxic compounds remains.
b. The potential to affect a local sensitive habitat or species.
c. Outstanding exposure issues where a risk assessment will allow realistic use of information about a species' natural history such as foraging areas, breeding times, migration patterns.

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d. Potential human health exposure either directly to sediments or through the food chain.

e. Issues associated with environmental windows (time periods when a species is least vulnerable).

The selection of personnel to conduct a risk assessment depends on the level of complexity addressed in the risk assessment. For example, a rough estimate of exposure based on a simple sediment-water partitioning equation may be sufficient to demonstrate little probability of bioavailability of a chemical, and hence risk. In such a case, operations personnel with expertise in engineering, chemistry, or marine geology may be the only necessary personnel. In the most complex assessments (and these are likely to be the least frequently encountered), an interdisciplinary team of engineers, biologists, chemists, and physical scientists may be necessary.